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The American Heart Journal

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The American Heart Journal

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Original Communications

ECONOMIC ASPECTS OF HEART DISEASE*

HAVEN EMERSON, M.D.
NEW YORK, N. Y.

NO SMALL part of the present colossal wealth of our country is due to the accumulated saving of the lives of men and women in the years of their greatest material productivity, by the fall in the death rate from pulmonary tuberculosis. It is unlikely that in this country and in our time any other change in the prevalence of disease can produce such impressive increase in the earnings and savings of our people. Now that tuberculosis has lost its leadership of the column of deaths and heart diseases have emerged, partly by the fall in the death rates from other causes and partly because of the great and sustained increase in the death rates from heart disorders as the most frequent of the causes of death, popular interest, as well as medical, drives us to analyze causes and costs in this field of pathology as a community problem.

In 1921 it was estimated that the *per capita* cost of tuberculosis to the people of the United States was \$7.96 annually, and that this burden, if distributed among those persons with an active, clinically recognizable tuberculosis, amounted to \$1,262 annually per patient, and that the total loss due to tuberculosis during the entire life span of the population at the then rate of tuberculosis mortality would amount to \$27,125,000,000.¹

No better confirmation of the adage, "The first wealth is health," is to be found than the estimate of economists that the value of human lives in this country is at least five times that of all material possessions.²

While the implications of the title of this paper may be as broad as the range of human activities, it will be understood for present purposes that "economics, or political economy, is the social science which treats of that portion of human activity which is concerned with earning a living," and that under the term heart disease are included those various conditions provided for under the four titles

*Presented before the Congress of American Physicians at Washington, D. C., May 2, 1928.

of the International List of Causes of Death, 87-90—Pericarditis, Endo- and Myocarditis (acute), Angina Pectoris, and Other Diseases of the Heart.

We are necessarily limited to the use of these few terms, inadequate as they are, because with rare exceptions under no others are there to be found records of the distribution and duration of heart diseases which permit us to estimate the character and the extent of the influence of this particular group of disabilities upon earning capacity and span of life.

The bearing of disease upon the earning of a livelihood depends at least upon the age of the person at onset, the degree and duration of disability as the process develops, and the extent to which death from this cause shortens life.

Information is therefore to be sought from the records of deaths, and of sickness, where these latter are assembled through institutions or agencies, such as hospitals, dispensaries, visiting nurse associations, industrial and insurance or compensation offices.

While no economist or statistician can express in figures alone the value of man's life, there is a trustworthy method of calculating in terms of earnings, the relative returns on the investment of life when death comes to man at different ages.

The studies of Dublin and Lotka² will be used as the basis of estimating the economic losses due to heart disease in this paper. By these authors the money value of a life at a given age is considered to be the difference between the person's calculated future earnings at the time of his death, and the present worth of his probable future expenditures.

Since the overwhelming majority of men in the United States today are found in, or at least nearer the \$2,500 income class of earnings than in the \$5,000 a year class or over, we shall confine our estimates to the former without attempting to make allowance for that relatively very small proportion of heart patients and deaths occurring in the higher income group. For convenience of reference, however, the accompanying abbreviated table from the studies of Dublin and Lotka will indicate the relative values of these two groups of earners expressed in dollars of value at the age of death.

ESTIMATED VALUE OF A MAN'S LIFE IN TERMS OF NET FUTURE EARNINGS AT VARIOUS AGES

| <i>Year of Age</i> | <i>On Basis of \$5,000 Annual Earnings</i> | <i>On Basis of \$2,500 Annual Earnings</i> |
|--------------------|--|--|
| 18 | \$34,321 | \$28,654 |
| 21 | 39,176 | 30,818 |
| 30 | 48,562 | 31,038 |
| 40 | 45,670 | 25,795 |
| 50 | 30,354 | 17,510 |
| 60 | 10,256 | 8,499 |
| 70 | 5,988 | 562 |

Our knowledge of the degree of disability due to heart diseases during the various phases of their preclinical and recognized existence in man is too fragmentary to justify us in attempting calculations in this field. For persons completing the years of their normal life existence; i.e., with undiminished average length of life, though handicapped because of limitations in physical vigor, skill, or endurance because of disease, reliable estimates of money value at the various ages can be made as has been indicated by Dublin in his table on Substandard Wage Earners in Three Income Classes (\$500, \$1000, and \$1500).³

The burden of heart diseases caused by the shortening of the life of occupied and retired civilians, from twenty to sixty-five years of age,⁴ is heavier among the unskilled workers than in any other social class, the unskilled workers' comparative mortality figure from this cause being 156.5, while that of the upper and middle class is 105.8 and that of skilled workers is 120.1, a difference to the disadvantage of the low income level group which is found also in tuberculosis, cancer, and pneumonia.

This difference is particularly marked when the death rates from heart diseases by social classes are analyzed by age groups, the unskilled workers suffering out of proportion in all decades of life, but particularly in those under sixty-five years, as shown in the accompanying list.

| <i>Age</i> | <i>Unskilled Workers</i> | <i>Skilled Workers</i> | <i>Upper and Middle Class Workers</i> |
|------------|--------------------------|------------------------|---|
| 16 - 19 | 25 | 18 | 6 |
| 20 - 24 | 34 | 25 | 15 |
| 25 - 34 | 55 | 37 | 18 |
| 35 - 44 | 91 | 61 | 43 |
| 45 - 54 | 181 | 127 | 112 |
| 55 - 64 | 454 | 394 | 401 |
| 65 - 69 | 946 | 907 | 977 |

As the Registrar General remarks, "It is evident that in early and middle life at least, heart disease as a whole is found to be especially fatal to the poorer classes, and although no record has been obtained of the social incidence of mortality from acute rheumatism, it is to be presumed that even if all classes were equally subjectable to this disease its effects upon the valves of the heart would be more serious to those least in a position to take the necessary precautions."

When the mortality from valvular disease of the heart is distributed by specific occupations, the death rates from this condition are found to vary widely according to the character of employment.

If we consider the death rate from valvular heart disease of all occupied and retired males (twenty to sixty-five years) as 1,000 we find the low mortality rates among,

| | |
|------------------------|-----|
| Insurance officials | 230 |
| Gamekeepers | 249 |
| Bank officials | 334 |
| Anglican clergy | 391 |
| Millers | 393 |
| Medical practitioners | 438 |
| Roman Catholic priests | 472 |

Those occupations with a high ratio of mortality from this cause are:

| | |
|-------------------------------|------|
| Cotton carders | 2170 |
| Cotton strippers and grinders | 2063 |
| Barmen | 2024 |
| Slate masons | 1994 |
| File cutters | 1852 |
| Cutlery grinders | 1727 |
| Slaters and tilers | 1727 |
| Cotton blowroom operatives | 1688 |
| Wool weavers | 1666 |
| Slate miners | 1647 |

Similarly the occupations showing particularly high mortality rates from "Other heart diseases" (chiefly myocardial) include many low paid and disadvantageous employments.

| | |
|------------------------------------|------|
| Tin and copper miners below ground | 3244 |
| Cutlery grinders | 2834 |
| Slate masons | 2642 |
| Barristers | 2514 |
| Hat formers and plunkers | 2317 |
| Tin and copper miners | 2242 |
| China, kiln, and ovenmen | 2122 |
| Pottery dippers, glazers, etc. | 2108 |
| Cellarmen | 2029 |
| Cotton carders | 1869 |

From the less ample and conclusive evidence in this country we find, as given in Pedley's⁵ tables of proportionate mortality from principal diseases among ten occupations in New York City, 1924, that butchers, tailors, cigar makers, and shoemakers have death rates from heart diseases considerably higher than the rate for all the occupations listed as a group. Furthermore, it is seen from this same study that in 1920 the proportionate mortality for occupied males in the United States, ten years and over, was higher from heart diseases than from any of the other six leading causes of death.

The duration of heart disease as determined by various sickness surveys, reported by Frankel and Dublin (1915-17) in North Carolina, Pennsylvania, New York,⁶ and Massachusetts differs radically from the duration of all sicknesses taken as a group. Calculations based on these surveys justify the following statements:

One-quarter of all sicknesses lasting less than two weeks; 3.7 per cent of all heart disease lasting less than two weeks.

Fifty-four and eight-tenths per cent of all sicknesses lasting less than three months; 28.6 per cent of heart disease lasting less than three months.

Seventy per cent of all sicknesses lasting less than one year; 50.5 per cent of heart disease lasting less than one year.

Sixteen and eight-tenths per cent of all sicknesses lasting three years and over; 24.2 per cent of heart diseases lasting three years and over.

Among New York State factory workers in 1919,⁷ 1.2 per cent of total wages were lost on account of heart diseases, or \$66.11 lost per case, while 2.2 per cent were lost on account of tuberculosis, \$230.51 lost per case.

Sickness from organic heart disease among office employees in a large commercial office⁸ in New York City caused a loss of two hundred fifty-three and nine-tenths working days per annum per 1000 persons employed, and showed a loss of ninety and six-tenths days per case of illness which constituted 2.68 per cent of all working days lost, the differences between male and female workers being of no significance. There were lost on account of heart diseases 296.8 calendar days for each 1000 persons employed, or 105.8 calendar days per case of heart disease, or 2.78 per cent of all calendar days lost on account of sickness among these employees. There were 2.8 cases of heart disease per 1000 persons employed in 1925 amounting to 3.4 cases per 1000 clerk years or 0.13 per cent of all cases of disability for that year.⁸

Analysis of health insurance claims on account of heart diseases⁹ shows that ninety and four-tenths days are lost per claim, varying according to the age of the claimant from forty-two days among those twenty-five to twenty-nine years of age to one hundred twenty-three and one hundred fifty days among those fifty to fifty-four and fifty-five to sixty years of age respectively.

In Brundage's study of *Sickness Among Industrial Employees*¹⁰ he found that according to the length of the maximum disability period provided for, the number of days of disability per case varied from sixty-five to one hundred twenty-two days, as compared with the duration of disability per case of eighty-four to one hundred sixty-one days in tuberculosis.

In the same report the average annual number of cases of heart disease as a cause of sickness disability per 1000 male industrial workers (1922-24) was found to vary from 1.1 in the public utilities to 1.4 among the iron and steel workers, and 1.5 in other industries.

In Dublin's study of *Causes of Death by Occupation* from the Metropolitan Life Insurance Company mortality experience,¹¹ deaths from organic heart disease constituted 12 per cent of deaths from all causes varying from 5.4 per cent among railway enginemen and trainmen to 16.5 per cent among farmers and farm laborers and for females 14.8 per cent, ranging from 8.1 per cent among bookkeepers and office assistants to 15.3 per cent among housewives and housekeepers. These differences are doubtless due in large measure to the small number of cases dealt with as well as to the age groups of the persons involved.

A further analysis of Dublin's study reveals for all occupations the percentages of all deaths represented by those due to heart diseases, according to the sex and decade of life of the deceased as shown in the accompanying age groups:

| | AGE GROUPS | | | | | |
|---------|------------|-------|-------|-------|-------|-------------|
| | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65 AND OVER |
| Males | 5.8% | 5.4% | 7.7% | 11.1% | 15.9% | 20.4% |
| Females | 6.9% | 7.0% | 10.0% | 13.9% | 18.7% | 20.8% |

Similar information upon percentage of all deaths represented by deaths from heart disease and decade of death is found in Wynne and Guilfoyl's¹² report for experience among employed males in New York City, 1914, as here shown.

| | AGE GROUPS | | | | | |
|--|------------|-------|-------|-------|-------|-------------|
| | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65 AND OVER |
| | 8.4% | 8.7% | 10.3% | 17.6% | 22.2% | 28.0% |

In both these reports there are considerable differences according to sex and occupation, but the variations are quite consistent among the age groups.

Regardless of age and sex, the variations in the percentage which heart disease deaths constitute of all deaths, by occupations, were found by Wynne and Guilfoyl to be as follows:

| | Per Cent |
|----------------------------------|----------|
| All occupied persons (53,541) | 17.9 |
| Blacksmiths | 15.8 |
| Cigar makers and tobacco workers | 18.9 |
| Clerks and bookkeepers | 14.0 |
| Compositors and printers | 13.6 |
| Garment workers | 20.7 |
| Laborers | 13.4 |
| Machinists | 14.1 |
| Painters and paper hangers | 14.9 |
| Railroad track and yard workers | 17.6 |
| Saloonkeepers and bartenders | 10.1 |
| Teamsters and drivers | 10.3 |

In Brundage's study of a ten-year experience with absences from work in the Edison Illuminating Company of Boston he found that there were for both men and women, 4 absences for heart diseases among each 1000 on the payroll per annum, but that while the men lost forty-nine and fifty-two hundredths calendar days on account of each such sickness, the women lost ninety-nine and seventy-three hundredths days.

Of the total deaths from all causes in the United States Registration Area, 1925, 1,219,019, there were 191,226, or 15.7 per cent, from heart diseases. Of these (215 age unknown), 170,482, or 89.2 per cent, were in persons forty years of age or over.

| | |
|----------------------|--------|
| 40 - 49 years of age | 16,384 |
| 50 - 59 | 28,118 |
| 60 - 69 | 44,843 |
| 70 - 79 | 49,774 |
| 80 - 89 | 27,262 |
| 90 and over | 3,886 |
| Unknown | 215 |

There were only 20,744 deaths from heart diseases in persons under forty years of age.

| | |
|-----------------------|--------|
| Under 10 years of age | 3133 |
| 10 - 19 | 4095 |
| 20 - 29 | 4879 |
| 30 - 39 | 8637 |
| | <hr/> |
| | 20,744 |

The decade seventy to seventy-nine years in which the largest number of deaths occurred is the last one for which Dublin estimates that there is any balance of money value in favor of the individual between his probable earnings and expenditures.

The largest number of deaths from nephritis and from cerebral hemorrhage also occur in the decade of life seventy to seventy-nine years, while the largest number of deaths from cancer occur in the decade sixty to sixty-nine and of tuberculosis in the decade twenty to twenty-nine years.

Not alone the total number of deaths from a particular disease, but their distribution among the decades of life, and the duration of the disease as a cause of relative or complete disability are the determining factors of its economic cost to the individual and the community.

In dealing with heart disease we must consider duration and death, moreover, on the basis of the three dominant etiological groups, rheumatic, syphilitic, and arteriosclerotic or senescent.

In translating clinical histories into graphic form for the purpose of expressing the spread of disability and death across the years of life, we have the following pictures of the three groups mentioned. (See Fig. 1.)

When rheumatism completes its cycle from onset through a period of disabling symptoms to and through the time of decompensation to death, all under the age of forty, which occurs in the great majority of cases, we find that the onset is under ten years of age in 19 per cent of the cases, between ten and twenty years of age in 37 per cent, between twenty and thirty in 23 per cent, and between thirty and forty in 21 per cent of the cases. Furthermore, we find on the average, a duration of four years between onset and partially disabling symptoms, which thereafter continue about seven years before decompensation, and with it probably complete disability develops, which continues, alternating with partial and occasional working ability, for four years until death.

With syphilis, the infection which occurs between eighteen and thirty years of age is followed, so far as heart disease including aortitis is concerned, by from ten to twenty-five years without symptoms or conscious disability, death following usually within two years after the development of disabling symptoms. The deaths from syphilitic heart disease are therefore to be found in 80 per cent of the cases after the age of forty, in 12 per cent in persons over sixty, and in about 34 per cent of the cases each in the decades forty to forty-nine and fifty to fifty-nine years.

Of the 20 per cent of syphilitic cardiac deaths occurring under forty years of age, 3 out of 4 are in the decade between thirty and forty years.

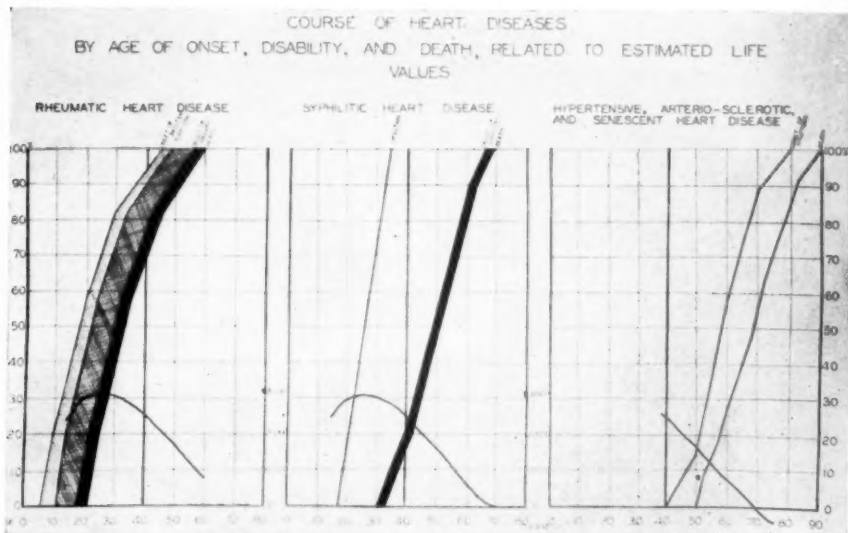


Fig. 1.—In this figure will be found graphically expressed the life history of the average heart patient of each of the three chief categories of heart disease across the span of life (summarized from the literature by Cohen¹³).

The onset of rheumatic heart disease occurs in 60 per cent of the cases before the age of twenty years and in 95 per cent before the age of forty. The period of time between infection and the development of disabling symptoms is usually longer in those attacked early in life than in those attacked after twenty, as shown by the varying width of the lightly shaded strip averaging a little less than four years in duration.

Following this is a fairly uniform period of seven years (heavily shaded strip) during which there are disabling symptoms of varying severity but causing interference to marked degree, though intermittently with usual occupations.

Finally, the solid black strip expresses the period of about four years of increasing and almost continuously complete disability until death results from cardiac failure.

The curved line A represents the changing value of a life expressed in dollars, throughout the period of prevalence of rheumatic heart disease as indicated at the right hand of the graph.

The same use of lines and spaces prevails in the graphs of experience with syphilitic and hypertensive or senescent heart disease.

In the great mass of all deaths attributed to diseases of the heart, those of the senescent type, constituting 90 per cent of the total, the onset of symptoms occurs (according to Wyckoff¹⁴) by decades as here shown:

| <i>Age</i> | <i>Per Cent</i> |
|-------------|-----------------|
| 30 - 39 | 0.4 |
| 40 - 49 | 14.1 |
| 50 - 59 | 43.6 |
| 60 - 69 | 31.6 |
| 70 and over | 10.2 |

This type of heart disease is not accompanied by any more, or other disability than is commonly found among all persons in the later decades of life, the subjective symptoms of the disease rarely causing the patient serious occupational handicap unless he be alarmed by what he is told about his sickness.

In most cases in the senescent type of the disease, cardiac failure of a disabling degree occurs, if at all, so near to the time of death and at so late an age that a calculable period of economic disability does not develop.

Practically all deaths attributed to the senescent type of heart disease occur after the age of fifty, their distribution among the subsequent decades of life being as shown in the accompanying table.

PERCENTAGE OF ALL DEATHS FROM SENESCENT HEART DISEASE FALLING IN THE DECADES

| <i>Years</i> | <i>Per Cent</i> |
|--------------|-----------------|
| 50 - 59 | 18 |
| 60 - 69 | 29 |
| 70 - 79 | 33 |
| 80 and over | 20 |
| | <hr/> 100 |

The following are typical composite histories from patients among the low income or dependent class, representing the three groups of heart disease leading to death, as assembled from the use of the detailed record forms, issued by the New York Heart Association.

Rheumatic Heart Disease.—White male patient, born in the United States of Italian parents. Reported at clinic at age of sixteen years. Patient had had rheumatic fever at the age of eleven and again at fourteen. At the age of fourteen he began to have symptoms referable to his heart, dyspnea, and precordial pain. A tonsillectomy was performed at the age of fifteen. When patient was seen in the clinic at age of sixteen, the diagnosis of mitral insufficiency and stenosis was made. At the age of eighteen he had his first attack of cardiac failure, with symptoms of dyspnea, orthopnea, and precordial pain. He was cyanotic and his liver was palpable. The following year, at the age of nineteen, he had another attack of failure, more severe than the first. The patient died at the age of twenty-one of progressive congestive heart failure.

Syphilitic Heart Disease.—Male patient was admitted to the clinic at the age of forty-nine years. He had had gonorrhea at the age of twenty-three and syphilis at the age of twenty-four. At the age of forty-eight he began to have cardiac symptoms, which progressed markedly until he died at the age of fifty.

Arteriosclerotic Heart Disease.—Male patient was admitted to the clinic at the age of fifty-five years. At the age of forty-nine he began to have symptoms of heart failure but not of a degree of severity to interfere with work. The disabilities progressed until the time of his death, at the age of fifty-seven.

Estimates of the cost of heart disease in the United States can be approximated by calculating the losses due to premature deaths, and the costs of medical and nursing care, as in hospitals, dispensaries, by visiting nurses, in convalescent homes, and in homes and hospitals for chronic invalids.

Death Costs, 1928.—The population of the United States is estimated as of July 1, 1928, to be a little over 120,000,000. The death rate from heart diseases (87-90) in the Registration Area in 1925 was 185.46 per 100,000 of the population.* It is estimated that the deaths from heart diseases in 1928, if the rate of 1925 is not exceeded, will be 221,809. When distributed by age groups according to the distribution in per cent of heart deaths in 1925 and charging for the numbers occurring at each decade, the value of a life at the mid-period of the decade, we find the loss in terms of money value to be \$1,538,897,680, or \$12.65 per capita of the population.

| Age Group | Estimated Distribution of Deaths From Heart Disease 1928 | | Life Value at Mid-period Age | | Loss to the Community |
|--------------|--|---------|---------------------------------|---------------|--------------------------|
| | Per Cent | No. | | | |
| Under 10 yr. | 1.6384 | 3,561 | \$14,156 | | \$ 51,598,620 |
| 10 - 19 | 2.1414 | 4,673 | 25,341 | | 120,724,524 |
| 20 - 29 | 2.5514 | 5,564 | 31,900 | | 181,064,400 |
| 30 - 39 | 4.5166 | 10,015 | 28,750 | | 288,851,250 |
| 40 - 49 | 8.5679 | 18,917 | 22,000 | | 419,298,000 |
| 50 - 59 | 14.7041 | 32,493 | 12,900 | | 421,946,100 |
| 60 - 69 | 23.4503 | 52,200 | 4,400 | | 229,530,400 |
| | | | | | \$1,713,013,294 |
| 70 - 74 | 13.4982 | 30,027 | - 766 | \$ 23,000,682 | |
| 75 and over | 28.9317 | 64,359 | - 2,348 | 151,114,932 | |
| | | | | | - 174,115,614 |
| | 100.000 | 221,809 | | | \$1,538,897,680 |

While it may be quite justified from a purely financial point of view to calculate the loss to the community of the life of a woman on the basis of 50 per cent less monetary value than that of a man at the same age, there is a good reason to consider that the loss of life from heart diseases among women, decade by decade, and for equal numbers, constitutes quite as heavy a financial loss to the family and home in terms of dollars' worth of service and care, as results from the same cause in the male. This is particularly the case in the economic group we are using as the basis of our estimates; i.e., persons earning \$2,500 a year or less. Therefore, no attempt will be made to proportion the total cost of heart disease, sickness, and deaths according to sex incidence.

*This death rate was 199.1 in 1926 and in all probability the rate for 1928 will be well over 200, but since the other data to be referred to is most of it available for periods not later than 1925, the calculations can more conservatively be based on the 1925 death rate of 185.46.

The death rate from heart disease in the registration area¹⁵ having increased 27.3 per cent from 1920 to 1925 (31 per cent among males, 18 per cent among females), it is certain that the 1928 estimate of deaths from this cause is conservative and well within the truth.

Hospital Costs.—In 1927 in the 4322 general hospitals in the United States, 66 per cent of the 345,364 beds were used on the average throughout the year, or 227,940. It was found¹⁶ in 1921, in New York City, that while 4.6 per cent of all general hospital patients are admitted for heart disease, 9.1 per cent of the days of care provided in these hospitals are for this class of patients, or a total of 7,562,837 days of care for heart patients in the United States or, at an average cost of \$4.00 a day per patient, a cost of \$30,251,348. The average cost of hospital care in public general hospitals in New York State has in recent years (1926) been \$3.08 per patient day, and in private or so-called endowed general hospitals \$4.90 per patient day.

The experience of the State of New York including the City of New York shows a much smaller proportion of all hospital patients to be heart patients (1926¹⁷); i.e., 2.7 per cent in the general hospitals of the State and City as compared with 4.6 per cent in general hospitals in the city taken separately. If then the 2.7 per cent of all general hospital patients in New York State and City are heart patients, and we have learned (1926) that the heart patient on the average uses about 1.74 times as many days of hospital care as other patients do, we shall find when applying this situation to the hospitals of the country as a whole that there would be 4.698 per cent of total bed days of hospital use devoted to care of heart patients, or 3,904,319 days ($2.7 \times 1.74 \times 227,940 \times 365$) which at four dollars a day of care would be \$15,617,276. The actual cost of hospital care for heart patients for the United States as a whole probably falls between these two figures; i.e., not over thirty nor under fifteen million dollars a year.

The reader familiar with the social practice of different parts of the United States as it affects the supply, use and *per diem* cost of hospital bed care for heart and other general medical patients will realize that estimates based on the experience in New York City and State may be in error, but information on the points at issue are not available from other large population groups. Also the fact that the reported death rate from heart diseases for New York State is higher than for any other state may result in an extent of hospitalization of this group of diseases above the average for the country as a whole. When more complete information is available, the estimates offered here and now will have to be revised, but if present trends in death rates and hospital use and costs continue, the revision is likely to be upward rather than down.

It was found, furthermore, that 7.4 per cent of the total cost of operating the public general hospitals in New York State, including New

York City, in 1926 went for the care of heart patients, and 3.6 per cent of the cost in the private general hospitals went for this purpose, the latter not being provided so commonly with beds available for the more chronic forms of disease. In the private hospitals in New York City the comparable figure was 4.3 per cent.

Nursing Visit Costs.—Approximately 3,000,000 persons are in the boroughs of Manhattan, Bronx, and Richmond of New York City, within the reach of the Visiting Nurse Service (The Henry Street Nurses). Among these people 304,367 nursing visits were made to patients discharged from care in 1927, of which 7,229, or 2.4 per cent, were for heart patients. Stated in another way these nurses visited 44,216 different patients in the year, of whom 692, or 1.6 per cent, were heart patients, who received an average of 10.45 nursing visits each.

Among the entire population of the United States, approximately forty times the size of the community served by these visiting nurses, either visiting nurses or other persons with less skill and at more expense directly or indirectly, will be found to be providing some degree of home nursing, or a total of 289,160 individual services, which at the present actual average cost of visiting nurse service of \$1.00 a visit, would be \$289,160.

Cost of Clinic Care.—The six million people of New York City appear to be adequately served by the several heart clinics, as far as heart patients, suitable on economical and pathological basis for ambulatory clinic care, are concerned. Almost all ambulatory heart patients not under the care of private physicians go direct or by reference from other out-patient or dispensary classes to the special heart clinics scattered throughout the city.

The patients attending these heart clinics numbered 10,017 (4592 adults, 5425 children) and made 37,871 separate visits.¹⁸ Of the total number of patients 22 adults and 211 children were found not to be suffering from heart disease.

Thus we find that there was one heart patient attending a heart clinic in the year for each 600 persons in the population and one clinic visit was made in the interest of heart disease by these patients for each 150 persons in the population.

It is known that as many as one-fourth of the entire population of the city calls upon the dispensaries for medical care in a year. We may reasonably estimate that there are four times as many ambulatory heart patients in the city receiving some medical care (three-fourths of them presumably at their own expense) as there are patients attending heart clinics.

Applying this ratio we should find that one person in 150 of the population of the country is a heart patient (800,000) capable of visiting a physician's private office or a public clinic, and that there is

made one visit by these heart patients each year for each forty persons of the entire population.

At this ratio we should have about 3,000,000 visits a year, those at the public dispensaries costing the public and the patients about \$1.00 a visit to provide, and the rest costing \$2.00 or more for each visit, or approximately \$750,000 for the dispensary visits and \$4,450,000 for private physicians' care or \$5,200,000 for the population of the country as a whole.

The possible error of too high estimates based on experience in New York City is recognized, but final correction on the basis of morbidity records on a national basis must wait for the distant future. It must be remembered that the American heart movement had its origin in New York City and the more the knowledge of prevalence of heart disease is developed elsewhere the nearer do other communities approach the records of New York.

Convalescent Care Costs.—We have estimated (on the basis of New York City's experience) that there may be 7,562,837 days of hospital care given to heart patients throughout the United States in a year, and about half as many if we base our calculations upon use of beds for heart patients in hospitals throughout the state outside of New York City.

In 1923 it was found¹⁶ that in large general hospitals of New York City the average length of stay of heart patients was fifty-two and two-tenths days for each such patient admitted, the length of stay varying from twenty days at Bellevue Hospital to one hundred and forty days at the City Hospital for chronic invalids.

It was found in 1927¹⁷ that the average length of stay of heart patients in 101 general hospitals in the state of New York was twenty-four days, varying from fourteen and three-tenths days in Westchester County to thirty-three days in Erie County, New York County giving twenty-six and three-tenths. Taking the more conservative and probably more representative figure of twenty-four days' care per patient, we can estimate on that basis that there were somewhere between 160,000 and 315,000 heart patients admitted to general hospitals according to our use of state or city figures for the number of beds occupied by heart patients in general hospitals.

It has been found in various surveys of hospital and dispensary practice and from studies of the needs of patients as discharged from hospitals¹⁸ that among general medical and surgical patients and maternity patients about 10 per cent needed as much as three weeks' convalescent care. More nearly twice as many of hospital heart patients require such a convalescent period as do the general run of patients, and for a six weeks' period instead of three weeks.

Similarly it is found that, while of general dispensary patients some 2 per cent need convalescent care for three weeks, probably at least

4 per cent of heart patients attending clinics need such care for as much as six weeks.

Twenty per cent of hospital heart patients would be somewhere between 32,000 and 63,000 according to our basis of estimating heart patients as above. Four per cent of the probable one-fourth of the ambulatory heart patients attending clinics (200,000 as given above) would be 8,000. Six weeks' convalescent care for these two groups of patients would amount to somewhere between 1,680,000 and 2,982,000 days of convalescent care.

This type of care costs about half as much *per diem* as does general hospital care, so the expense of such care if provided would be between \$3,360,000 and \$5,964,000.

We do not know the number of days of care provided for heart patients in convalescent homes, but we do know²⁰ (Bryant, *Convalescence*, 1927, pp. 255-257) that there are about 12,000 beds devoted to convalescent care for part or all of the year outside of the vicinity of New York City and about 3,000 more in the New York City environs. Many of these beds are available for only a few months each summer. Others, though intended for use throughout the year, are not always occupied, and there is no reliable record of the number of bed days of convalescent care provided for heart patients.

However, since New York City's 6,000,000 people are pretty well served in respect to convalescent care for heart patients by the 370 beds in use for this purpose throughout the year and used to a high percentage of their capacity, we might expect twenty times as many beds to be needed for the country as a whole or 7,400 which falls between the figures given above, 1,680,000 days of care calling for 4602 beds and 2,980,000 days requiring 8164 beds. Our estimates of cost are then within reasonable limits.

It is fully recognized that no such amount of care for convalescent heart patients is at present provided for in institutions operated for this purpose. However, if heart patients do not have such care in institutions or its equivalent under individual conditions, they will suffer avoidable damage from lack of it. Therefore, this item is added to the total estimate as a proper charge.

Chronic Sick Costs.—In a survey of chronic sickness, recently completed in Boston,²¹ it was found that one person among every 185 of the population was a chronic invalid and that 19.6 per cent of these were heart patients, of whom 30.7 per cent were persons under twenty years of age and 42.01 per cent were persons under forty years of age. All but 30 per cent of all chronic heart patients were found to need care throughout the year, and it was further found that this was generally provided at about one-half the cost per patient day of the cost of care in general hospitals.

On this basis applied to the United States as a whole, the estimated 89,122 chronic heart patients needing care the year round are probably costing in the neighborhood of \$65,059,060.00 a year. ($120,000,000 \div 185 \times 19.5$ per cent $\times 70$ per cent = 89,122. $89,122 \times 365 \times \$2.00 = \$65,059,060.00$.)

Assembling our variously estimated items of the cost of the medical and nursing care of heart patients in the United States in this year, we have in round numbers for those:

| | |
|---|-------------------------------------|
| A. Under care in general hospitals | \$15,617,000 to \$ 30,251,000 |
| B. Under care by nursing visits in homes | 289,000 |
| C. Attending clinics or physicians' offices | 5,200,000 |
| D. Under care in convalescence | 3,360,000 to 5,964,000 |
| E. Under care in chronic stage | 65,059,000 |
| Total cost for a year | <hr/> \$89,525,000 to \$106,763,000 |

We might properly say that we have estimated the above costs on the basis of a total of 1,148,800 to 1,179,800 heart patients of the various categories in a current given year or just under 1 per cent of the population as follows:

| | |
|---|------------------------------|
| A. Under care in general hospitals | 160,000 to 315,000 |
| B. Under care by nursing visits in homes | 27,680 |
| C. Attending clinics or physicians' offices | 800,000 |
| D. Under care in convalescence | 232,000 to 263,000 |
| E. Under care in chronic stage | 89,120 |
| | <hr/> 1,148,800 to 1,179,800 |

Estimates have ranged from 1 to 2 per cent of the population as constituting the total of heart patients.

Using 1 per cent of the population as being nearer our own estimate, built up from the various groups probably receiving care, and using the lower of our total cost figures (\$89,525,000), we find that the average heart patient in the United States today probably carries an annual charge of \$74.60 or we might properly say that heart disease puts a burden of about 75 cents on each person of the entire population each year.

The cost of heart disease deaths occurring in the various decades of life was found to be \$1,538,897,680 each year, or a loss of \$12.82 for each member of the population.

In 1922 at the then death rate from tuberculosis in the United States, it was estimated¹ that the shortening of life by this disease, which amounted at that time to a reduction of the average length of life of all the people of the country by two and five-tenths years each, would cost the entire population of the country at that time during their complete life span the sum of \$27,125,000,000.

On the basis that in 1928 heart diseases will cause a reduction in the average length of life of people in the United States of one and sixty-six hundredths years for males and two years for females, the cost to

those now living in the United States from the shortening of lives by heart disease will amount to \$21,960,000,000 during their life span.

While heart diseases cause more deaths than tuberculosis, the age of death for the latter is much earlier, hence the greater effect of tuberculosis deaths on the average length of life, and on the cost of tuberculosis to the population as a whole.

It has been estimated recently by Louis I. Dublin²² that the loss each year, on the basis of 1927 experience, due to the shortening of life by cancer, amounts to about \$680,000,000 which may be compared with the estimates for the population of 1928 on the basis of the 1925 heart disease death rate, as presented above, \$1,502,198,355, the greater annual cost from heart disease deaths being due to their greater frequency in the population, even though the largest number of deaths from this cause in any one decade of life is in a later decade than is the case with cancer deaths.

It was estimated on the experience of 1922 in the United States that the annual cost of tuberculosis patients was probably about \$3.15 per capita of the population. It has just been shown that for heart patients the entire population must bear a cost of 75 cents per capita per annum. This difference in the cost is probably due to at least two important factors: namely, the greater adequacy of provision for the medical and nursing supervision of tuberculosis as compared with those available for heart patients, and the added costs of sanitary supervision and isolation of many tuberculosis patients, of a kind not required for heart patients, except for those in certain stages of acute rheumatic fever and its recrudescences.

Probably some postponement in the age of death from heart diseases of rheumatic origin and possibly some reduction in the incidence of acute rheumatic fever might result from an expenditure per patient, or per capita of population, for heart disease more nearly equal to the sums found desirable for the prevention of tuberculosis and the treatment of the tuberculosis patient.

The burden of heart disease falls more heavily, absolutely in terms of incidence and deaths, and relatively in terms of loss of livelihood, upon the unskilled wage-earner, the under-privileged man and woman, than upon any of the higher earning or social levels of the community.

Because of its duration, heart disease, with the exception of tuberculosis, mental alienation, and certain forms of chronic arthritis, constitutes heavier burdens upon wage-earners than do other diseases, this burden increasing with each decade of life from twenty-five to sixty-five.

Grateful acknowledgment is here made for the valuable material collected and prepared for use in this paper by Miss Jessamine Whitney and Miss Beatrice A. Myers of the staff of the American Heart Association and by Miss Claire Lingg of the staff of the Heart Committee of the N. Y. Tuberculosis and Health Association.

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EXPERIMENTAL PERICARDITIS*†

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INTRODUCTION

IT IS a matter of common clinical experience to have unsuspected cases of chronic adhesive pericarditis come to light at autopsy. These are usually revealed without ever having been suspected, or, at least, without having been diagnosed or clinically proved during the life of the patient. In a pathologico-clinical study of all of the cases of pericarditis that came to autopsy in the Charity Hospital at New Orleans during a recent five-year period we¹ confirmed this general conception of the present state of our knowledge of the subject. We found that there were no pathognomonic signs of an adherent pericardium either with or without mediastinitis, but not infrequently one or more of the classical signs was present. The presence of any one of these signs, we concluded, should lead to a thorough clinical study to substantiate or disprove the suggested diagnosis.

Almost every physician has seen cases of unmistakable acute rheumatic or tuberculous pericarditis subside and heal without leaving a vestige of the previous process. Often not one of the classical signs is discoverable even on the closest, particularly directed scrutiny. Yet these patients may present clinically more or less persistent cardiac embarrassment, and at autopsy there may be revealed a complete or partial obliteration or synechia of the pericardial cavity, with or without mediastinitis. A carefully elicited history is usually of distinct importance. It is, however, all too frequently negative.

The multiplicity of infrequent and obscure physical signs of extrapericardial as well as intrapericardial adhesions is most confusing. The need is apparent for more reliable physical signs and graphic pathognomonic evidence for establishing the diagnosis of chronic adhesive pericarditis. No shadow of doubt as to the presence of pericardial adhesions should be permissible where any such heroic therapeutic measure as surgical intervention is contemplated. We know of at least one instance where a patient, apparently without adequate study and certainly on insufficient evidence, was put through an unnecessary pericardiectomy. At the operation a perfectly normal mediastinum and pericardial cavity were exposed. Such an error emphasizes the necessity for more absolute criteria than we have had in the

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past. The hope of finding further diagnostic data apparently lies only in the diligent prosecution of further extensive experimental and clinical studies.

THE PROBLEMS OF CHRONIC FIBROUS PERICARDIAL ADHESIONS

The effects upon the heart itself of the various types of chronic fibrous pericarditis, that is, the pathological myocardial response to the different grades and types of pericardial lesions and the extent of the functional impairment, are not definitely established. The factors that determine the myocardial response to chronic pericardial injury and the mechanism of the same are not yet known. In our pathological studies we have been struck with the variability in the degree of cardiac hypertrophy and the rarity with which atrophy of the heart was found. The reasons for these variations have intrigued us.² We have calculated comparative heartweight-bodyweight ratios in the cases of dogs with various types of pericardial and mediastinal adhesions. We have turned to account the standards and methods that have been used in previous cardiac hypertrophy studies in the dog.³

The common type of chronic mediastinopericarditis is generally considered to cause an undue strain on the heart, especially when there is parietal chest wall anchorage. The precordial framework of ribs is frequently removed so as to relieve the heart from the tugging against a fixed, firm resistance. We are prone to consider this as little more than a first stage operation since the important intrapericardial adhesions remain.

A complete synechia without extrapericardial adhesions, it is quite generally argued, is of no serious moment and does not interfere in any way with cardiac function. With this conception we are not in accord. The lubricative function of the pericardium is perhaps of little significance. The presence, however, of dense adhesions in the interventricular grooves, where there is the least cardiac movement, is of serious moment since the major blood vessels traverse these grooves. Contracting adhesions, by constricting the vessels, inevitably interfere to some degree with the circulation of the heart itself. The pericardium is always pathologically altered by any inflammatory process, and its restraining function becomes significantly less effective.

Delorme⁴ in 1898 on the basis of one poorly selected case, argued that intrapericardial adhesions were a source of serious embarrassment to the heart, and he contended that separation of the parietal and visceral pericardium should be accomplished by open operation. His contentions, however, are usually discarded with the argument that the adhesions invariably reform. V. Schmieden⁵ has in recent years successfully dissected off the adherent pericardium from the ventricles, in human cases with cardiac failure, because of the embarrassing effect on the cardiac movements. The Brauer⁶ operation of cardiolysis,

which consists in the removal of the rigid points of fixation, has received much more attention. This operation relieves the embarrassed heart to some extent where external adhesions have been firm and short. Yet it is merely a palliative procedure and does not correct or remove the significant intrapericardial lesions. These latter, we believe, are more significant than they are generally considered to be.

THE EXPERIMENTAL STUDY

For the past three years we have been engaged in the production of experimental pericarditis and the study of the clinical and anatomical facts gathered from these experiments. We have operated upon 125 dogs, but because of many technical difficulties only about half of our experiments have yielded data of significance.

Methods.—Our routine program for each dog has been rather extensive. Control fluoroscopic studies and roentgenograms were made. Control electrocardiograms with the animal in each of the three standard positions, on the back, on the left and on the right side, used to determine the presence or absence and the degree of shifting of the electrical axis, were taken with the animal under barbital anesthesia. Pericardiotomy was then performed under complete general intratracheal ether anesthesia, exercising at all times rigorous surgical aseptic technic. Irritating inorganic or organic compounds were introduced into the pericardial sac to produce the pericarditis. The pericardium and pleura as well as the thoracic incision were carefully closed. Fluoroscopic examinations and roentgenograms were then made, and electrocardiograms in the three positions were taken at irregular intervals and always just before the re-operation or the sacrificing of the animal with chloroform.

The dog's body weight was recorded and the heart carefully removed after making notes of all external parietal and mediastinopericardial adhesions. The type and extent of the visceral or intrapericardial adhesions and the degree of obliteration of the pericardial cavity were noted. The major blood vessels were cut short; the heart was then washed free of blood, fixed, cleaned, and divided, at a later date, according to the standardized technic previously described.³ The weights and ratios determined were compared with the established normals.³

THE RESULTS FROM THE EXPERIMENTS

Clinical Data.—The significant data obtained from the series of experimental animals will be reported upon here in a summarized form. The clinical, or physical, examination of the dogs yielded little information of diagnostic importance. No neck vein phenomena could be seen in any of the animals even on careful inspection. Retractions of the precordial area were very infrequent and were conspicuous only in animals in which the pericardium had been securely sutured to the anterior chest wall at the time of the initial pericardiotomy. The dog's heart is more freely and more longitudinally suspended than is the human heart. The mediastinum, furthermore, is much less dense, less firm, and less rigid than is that of the human being. This may account for the lessened tendency to the production of adhesions which would cause systolic retraction. Systolic and diastolic shocks were often

palpable over the dog's precordium. Although these palpatory phenomena, especially the diastolic shocks, were common, they cannot be considered as pathognomonic signs. The pulse was frequently found to be of a distinct paradoxical type, but it was not invariably so and

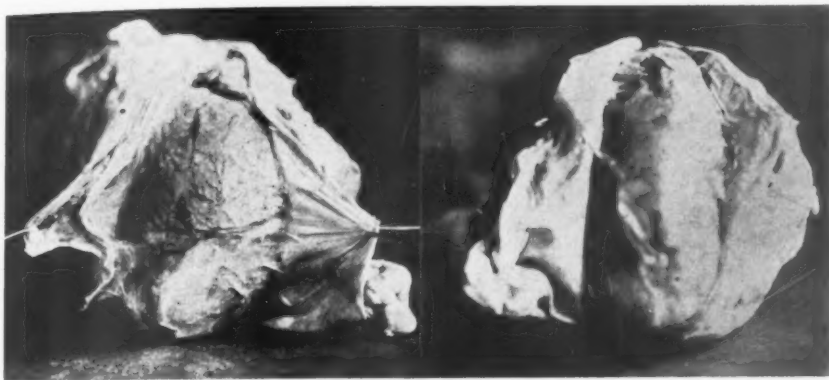


Fig. 1.—Chronic adhesive pericarditis. Anterior and posterior views of the excised heart of dog No. 115, with the pericardium slit and stripped back to show the firmness and extent of the adhesions between the parietal and visceral pericardium.



Fig. 2.—Chronic adhesive pericarditis. View of the heart of dog No. 185, in situ, with the pericardium split, dissected back, and pulled away by the five attached mosquito forceps.

the sign was absent in many dogs in which complete synechia of the pericardium was present (Figs. 1, 2, 3, 4).

Electrocardiographic findings (Fig. 5) were of definite significance only when fixation of the electrical axis in all three standard positions was positively established. The shifts, however, probably because of the position and suspension of the heart of the dog, are slight in some

normal dogs, and doubling the sensitivity of the string of the galvanometer is often necessary to demonstrate differences in the heights of the complexes. This in itself greatly distracts from the value of electrocardiographic studies under the experimental conditions. We

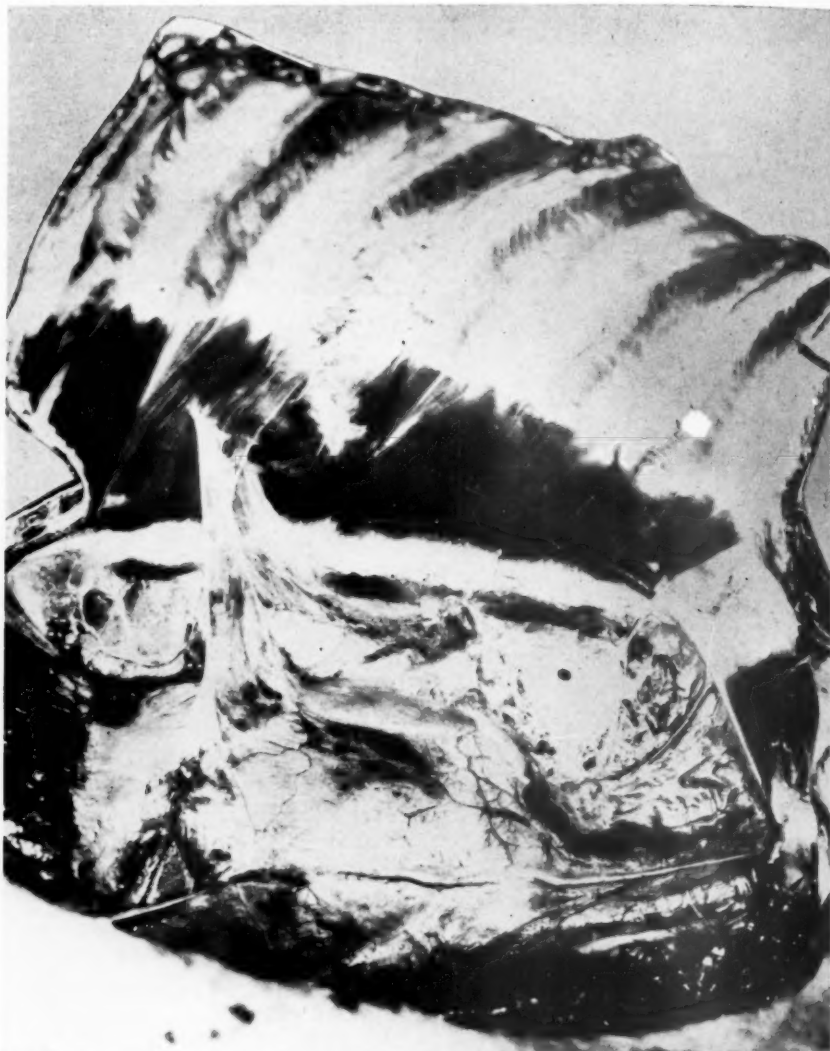


Fig. 3.—Chronic adhesive pericarditis and mediastinitis. Anterior mediastinum and heart in situ of dog No. 206, showing the extent of the mediastinal fibrosis and the parietal adhesions that the dogs in group IV had along with obliteration of the pericardial cavity.

were able to find definite fixation only when extrapericardial as well as intrapericardial adhesions were present. This is what one might expect. It was, however, common to find some anterior adhesions and complete synechia pericardii in dogs that had shown definite shifting

of the electrical axis. These discrepancies must be accounted for by the thinness of the dog's mediastinum and the peculiar suspension of the dog's heart with the long axis more nearly in a sagittal plane.

In our opinion, these discouraging results as to the value of the electrocardiographic sign of fixation of the electrical axis in adhesive pericarditis cannot be considered to hold true in the case of human beings. The suggestion may, however, be accepted that there is necessity for caution in the interpretation of the finding of fixation of the electrical axis described by Dieuaide and Carter as pathognomonic in itself of adherent pericardium. The diagnosis should not rest on the electrocardiographic sign only, and, of course, this would rarely be the



Fig. 4.—Chronic adhesive pericarditis without mediastitis. Transparent, filmy, diaphanous veil of the normal anterior mediastinum as it was preserved in the dogs of group V that otherwise had had complete pericardial obliteration experimentally produced.

case for usually there are other physical signs present which suggest the special electrocardiographic study in the three positions, for the routine procedure is to take the curves in one position only. It must be admitted, however, that the electrocardiographic findings in respect to the electrical axis are much more significant in humans. The human mediastinum, as is well known, is a denser, firmer, and more rigid tissue than the mediastinum of the dog, and the human heart is more transversely placed, rests on the diaphragm, and comes closer to the anterior chest wall.

The fluoroscopic studies yielded some interesting data, which the roentgenograms (Fig. 6) failed to show. We attempted to record

graphically, by means of moving pictures and by the roentgenographic slit method, the movement of the left heart border. Our attempts have been unsuccessful thus far. We have, however, noted a very definite change in the movements of the left cardiac border, especially in the region of the auriculoventricular groove where there is normally to be seen under the fluoroscope the see-saw movement with the fulcrum in the auriculoventricular groove at its extreme left end.

THE HEART WEIGHTS AND RATIOS IN THE EXPERIMENTAL ANIMALS WITH
ADHESIVE PERICARDITIS

Observations on Hypertrophy.—The body weight of the animal was recorded. After noting the gross external adhesions at autopsy, the heart was removed and freed of blood. The vessels were cut flush

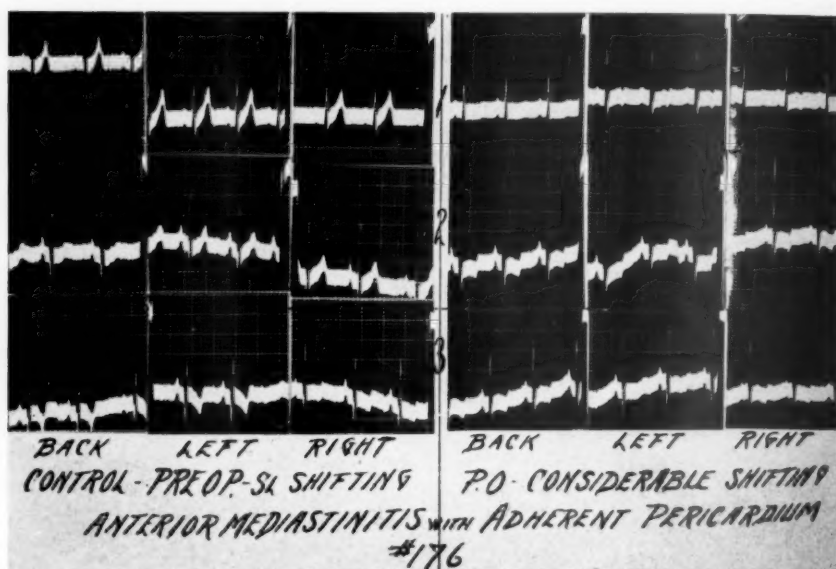


Fig. 5.—Electrocardiograms of dog No. 176 with the Leads I across the top, Leads II in the middle horizontal row, and Leads III in the bottom row. The first three columns to the left represent the three control leads, respectively, taken on the back, on the left side, and on the right side. Only slight shifting is present, as evidenced by the slight differences in heights of the R-waves in the three positions. The three columns to the right represent the curves taken after the production of a chronic mediastinopericarditis. It will be seen that the differences in heights of R-waves, especially in Lead I, are much greater, which indicates even increased shifting of the electrical axis, and, supposedly also, the anatomical axis.

with the auricles and the semilunar cusps and the heart fixed according to the standard formalin procedure.³ The kidneys and spleen were removed and their weights recorded.

The heart capacities and ventricular thicknesses were measured, and the heart was divided by the midseptal method and by Lewis' method. The ratios of the left ventricle weight to that of the right ventricle and

of the whole heart to the body weight were established. These figures are recorded in the accompanying tables, which will be here summarized.

Table I shows that there are practically no changes from the ratios for normal dogs in the values for the L/R ratio of the left ventricular weight to the right ventricular weight and of the total heart weight to the body weight (HW/BW) ratios of dogs with acute purulent pericarditis and mediastinitis lasting from eight to three days. This indi-

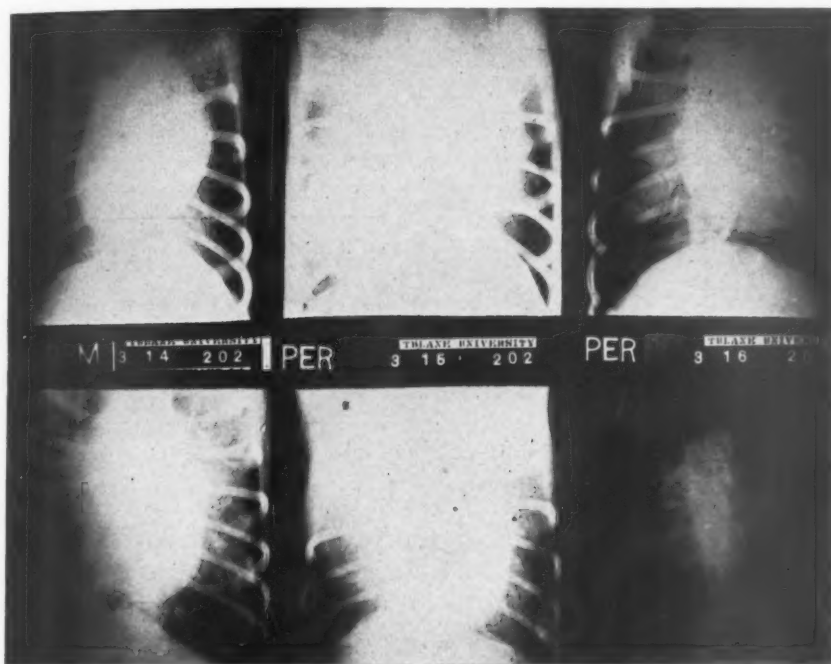


Fig. 6.—A short series of roentgenograms showing how the enlarged shadow of the acute pericardial effusion decreases and the gradual slight cardiac enlargement supersedes.

cates that these acute lesions for short periods are not associated with a response by hypertrophy on the part of the heart. These dogs make an excellent control series.

TABLE I
ACUTE PURULENT PERICARDITIS WITH ACUTE MEDIASTINITIS

| DAYS | L/R | HW/BW | DAYS | L/R | HW/BW |
|---------|-------|--------|------|-------|--------|
| 8 | 1.635 | 0.0099 | 3 | 1.105 | 0.0086 |
| 8 | 1.500 | 0.0081 | 3 | 1.088 | 0.0093 |
| 6 | 1.582 | 0.0088 | 3 | 1.037 | 0.0097 |
| 6 | 1.300 | 0.0085 | 3 | 1.092 | 0.0099 |
| 4 | 1.057 | 0.0089 | | | |
| Average | | | | 1.081 | 0.0090 |
| Normal | | | | 1.398 | 0.0080 |

Table II contains the heart data from 5 dogs in which there were external adhesions but no internal fibrous pericardial bands. These show no conspicuous changes from the normal except for a slight tendency to right ventricular preponderance. Apparently this type of lesion embarrasses the heart the least of any of the chronic lesions studied. The last two dogs were studied in conjunction with Dr. Alton Ochsner. These two dogs had had complete synechia, which was relieved by a secondary operation and the re-formation of the fibrous adhesions prevented. This was accomplished by the application of a digestant solution after cardiac decortication by digital separation of the adherent pericardial layers. The changes were not conspicuous.

TABLE II
CHRONIC ANTERIOR MEDIASTINITIS WITH THE EPICARDIUM UNINVOLVED

| NO. | | TOTAL DAYS | | L/R | HW/BW | |
|---------|-----------------|------------|-----------------|-------|--------|--------|
| 127 | | 516 | | 1.100 | 0.0085 | |
| 128 | | 200 | | 1.075 | 0.0092 | |
| 120 | | 31 | | 1.500 | 0.0092 | |
| 122 | | 11 | | 1.329 | 0.0091 | |
| | PO ₁ | | PO ₂ | | | |
| 169 | 107 | + | 14 | 121 | 1.210 | 0.0093 |
| 177 | 91 | + | 5 | 96 | 1.082 | 0.0097 |
| Average | | | | 1.216 | 0.0092 | |
| Normal | | | | 1.398 | 0.0080 | |

PO₁—Length of time after the first operation.
PO₂—Length of time after the second operation.

Table III shows the effect on the heart weights and ratios of anterior mediastinal adhesions associated with partial synechia of the pericardium. Under these conditions the dogs that had the lesions for more than eighty days showed distinct cardiac hypertrophy with a definite increase in the preponderance of the right ventricular weight. As a matter of fact, in the animals that lived long there was actually a larger right than left ventricle.

TABLE III
PARTIAL PERICARDIAL OBLITERATION WITH MEDIASTINITIS

| NO. | DAYS | L/R | HW/BW |
|---------|------|-------|--------|
| 124 | 502 | 0.885 | 0.0133 |
| 130 | 212 | 0.978 | 0.0100 |
| 147 | 108 | 1.000 | 0.0130 |
| 149 | 83 | 1.062 | 0.0131 |
| 132 | 81 | 1.360 | 0.0079 |
| 195 | 55 | 1.135 | 0.0078 |
| 125 | 43 | 1.223 | 0.0096 |
| 202 | 34 | 1.161 | 0.0084 |
| Average | | 1.101 | 0.0104 |
| Normal | | 1.398 | 0.0080 |

Table IV presents the heart weight ratios from dogs that had had complete synechia of the pericardium along with anterior medias-

tinitis. These dogs showed the greatest degree of general hypertrophy and also right ventricular preponderance. In all instances where the lesion had been present more than thirty-five days, definite evidence of hypertrophy was revealed.

TABLE IV
COMPLETE PERICARDIAL OBLITERATION WITH MEDIASTINITIS

| DAYS | L/R | HW/BW | DAYS | L/R | HW/BW |
|---------|-------|--------|------|-------|--------|
| 331 | 0.641 | 0.0155 | 42 | 1.144 | 0.1008 |
| 252 | 0.773 | 0.0204 | 37 | 1.180 | 0.0120 |
| 221 | 1.150 | 0.0133 | 35 | 1.000 | 0.0140 |
| 116 | 1.180 | 0.0117 | 33 | 1.078 | 0.0092 |
| 111 | 1.056 | 0.0114 | 33 | 0.963 | 0.0083 |
| 97 | 1.107 | 0.0091 | 17 | 1.070 | 0.0116 |
| 62 | 1.200 | 0.0103 | 16 | 1.250 | 0.0094 |
| 45 | 1.200 | 0.0100 | 13 | 1.058 | 0.0086 |
| Average | | | | 1.162 | 0.0116 |
| Normal | | | | 1.398 | 0.0080 |

Table V is made up of data from dogs that had had complete synechia of the pericardium (for varying periods of days) which embarrassment was relieved by a secondary operation done in association with Dr. Alton Ochsner, and the dogs sacrificed at intervals after the second operation. These animals showed less evidence of the cardiac response of hypertrophy. The results, however, though less conspicuous, still correspond in general to our findings in Table VI. The animals had for the most part relatively slight mediastinitis and in this way resembled those in Table VI.

TABLE V
COMPLETE PERICARDIAL OBLITERATION WITHOUT AND WITH SLIGHT MEDIASTINITIS,
ALL SECONDARILY OPERATED UPON

| NO. | PO ₁ | PO ₂ | TOTAL DAYS | L/R | HW/BW |
|---------|-----------------|-----------------|------------|-------|--------|
| 169+ | 107 + | 14 | 121 | 1.210 | 0.0093 |
| 177+ | 91 + | 5 | 96 | 1.082 | 0.0097 |
| 176 | 97 + | 10 | 107 | 1.200 | 0.0076 |
| 195 | 45 + | 10 | 55 | 1.134 | 0.0078 |
| 197 | 30 + | 18 | 48 | 1.150 | 0.0072 |
| 203 | 14 + | 21 | 35 | 1.040 | 0.0070 |
| 192 | 25 + | 7 | 32 | 1.099 | 0.0096 |
| Average | | | | 1.125 | 0.0085 |
| Normal | | | | 1.398 | 0.0080 |

PO₁—Length of time after the first operation.

PO₂—Length of time after the second operation.

Table VI contains the heart data of dogs that had had internal pericardial adhesions only for varying periods of days. It is of considerable significance inasmuch as the dogs in the series were operated upon in association with Dr. Alton Ochsner, whom we called upon for help in obtaining complete obliteration of the pericardial cavity without any mediastinitis whatsoever. The L/R and HW/BW ratios are

TABLE VI
COMPLETE PERICARDIAL OBLITERATION WITHOUT MEDIASTINITIS

| DAYS | L/R | HW/BW | DAYS | L/R | HW/BW |
|---------|-------|--------|------|-------|--------|
| 107 | 1.113 | 0.0086 | 34 | 1.048 | 0.0084 |
| 90 | 1.095 | 0.0123 | 28 | 1.030 | 0.0093 |
| 60 | 1.358 | 0.0120 | 25 | 0.912 | 0.0095 |
| 50 | 1.000 | 0.0120 | 25 | 1.142 | 0.0070 |
| 37 | 1.200 | 0.0105 | 14 | 0.955 | 0.0084 |
| 34 | 1.160 | 0.0084 | | | |
| Average | | | | 1.092 | 0.0097 |
| Normal | | | | 1.398 | 0.0080 |

similar to those of Table V and are interesting in that they show a distinctly less degree of cardiac hypertrophy and of right ventricular hypertrophy than we encountered when, in addition to the intrapericardial adhesions, there was also extrapericardial anchorage (Table IV). We did not find, however, as might be expected from clinical reports, cardiac atrophy.

The averages for the heart weight ratios of the various groups are brought together to facilitate comparison of the effects of the various experimental procedures.

COMPOSITE TABLE
EXPERIMENTAL PERICARDITIS

| | L/R | AVERAGE HW/BW |
|---|-------|------------------|
| I. Ac. Pur. Per. with Med. | 1.081 | 0.0178 |
| II. Chr. Ant. Med. without Per. | 1.216 | 0.0091 |
| III. Part. Per. Obl. with Med. | 1.101 | 0.0104 |
| IV. Comp. Per. Obl. with Med. | 1.162 | 0.0116 |
| V. Comp. Per. Obl. without Med. | 1.092 | 0.0097 |
| VI. Comp. Per. Obl. without and with Sl. Med.—Reoperated | 1.125 | 0.0085 |
| Normal | 1.398 | 0.0080 |

SUMMARY AND CONCLUSION

We have been able experimentally to reproduce at will in dogs the various types of chronic fibrous pericarditis which are encountered clinically.

The animals with chronic adherent pericarditis presented no pathognomonic clinical signs, except perhaps the localized systolic tugging in instances in which the parietal pericardium had been anchored to the intercostal muscles.

Electrocardiographic evidence of fixation of the electrical axis was, much to our disappointment, not uniformly present. This, however, may be due in part to peculiarities of the mediastinum and the midline suspension position of the heart in the dog.

After the production of an obliterative pericarditis fluoroscopic studies revealed a rather striking change in the character and movements

of the left heart border, especially in the region of the auriculoventricular junction. A graphic record of this changed movement may be of some diagnostic significance.

The gross pathological studies in regard to cardiac hypertrophy more or less confirmed our previous conceptions. The greatest degree of cardiac hypertrophy was found in the group in which chronic mediastinal and parietal adhesions as well as adhesive pericarditis were present. The tugging of the heart bound by fibrous bands from the firm structures of the chest is apparently most conducive to hypertrophy. The dogs with partial obliteration of the pericardial and mediastinal and parietal adhesions showed the second greatest degree of hypertrophy. Complete synechia of the pericardium with slight or with no mediastinal fibrosis showed hypertrophy but of less degree than the other types of chronic mediastinopericarditis.

The feasibility of digital separation of pericardial adhesions and the prevention of the re-formation of adhesions have been proved, and clinical and pathological evidences of benefit in the experimental animal have been noted.

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HEART-BLOCK SHOWING MULTIPLE TRANSITIONS ASSOCI-
ATED WITH CONVULSIVE SYNCOPE: REPORT
OF A CASE WITH DETAILED HISTO-
PATHOLOGICAL STUDY

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FROM time to time, in medical literature, cases of complete heart-block have been recorded in which have been demonstrated gross or histopathological lesions that interfere with or completely obstruct the passage of the impulse through the auriculo-ventricular bundle (His). The incidence of such cases is so low that the publication of additional cases is justified.

Lesions of various kinds have been described and notable among them have been gummas.^{2, 3, 4, 6, 9, 12, 13, 14, 21, 28, 34, 35, 40} Areas of calcification that encroach on or destroy the continuity of the bundle have been recorded by some observers.^{7, 8, 15, 17, 25, 29, 31, 38, 39} In other cases, obliterative disease of the branches of the coronary arteries which supply the region of the bundle has been described.^{10, 29} Fibrosis of the auriculo-ventricular bundle likewise has been observed.^{11, 16, 23, 36, 39} Fatty infiltration of the bundle, and also simple lymphocytic infiltration,¹⁷ occasionally have been noted.^{5, 30} In a few cases, infarction of the heart near, or in, the interventricular septum has resulted in complete heart-block.^{20, 27, 41} Involvement of the auriculo-ventricular bundle by ulceration associated with mural endocarditis has been noted.¹⁹ A few instances of tumors of the septum causing obstruction or destruction of the bundle have been reported. These tumors comprise endothelioma,¹ round-cell sarcoma,²⁶ and fibroma.³⁷ In a few cases in which complete heart-block existed, a lesion of the bundle was not demonstrated.^{18, 22, 32, 33}

The case which constitutes the basis of this report presented some extremely interesting and unusual features.

CASE REPORT

A man aged seventy-four years presented himself for examination because of fainting spells. His health up to three and a half years before had been good. Previous illness of all types was denied. He had two grown children, living and

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well. Three and a half years before, during a general examination which was conducted owing to the presence of an inguinal hernia, the systolic blood pressure was found to be 190 and the diastolic 110. Cardiovascular symptoms were not elicited, and the patient was active for his age. Three months before the last examination he began to have dizzy spells, occasionally associated with momentary unconsciousness. These attacks gradually increased in frequency until the time of his visit to The Mayo Clinic; their average occurrence was two or three times a day.

The heart was enlarged; the total area of dullness was about 16 cm. The rate was slow, only 40 each minute. The aortic second sound was accentuated. The peripheral arteries were distinctly palpable and sclerotic. The systolic blood pressure was 240; the diastolic, 105. The lungs were slightly emphysematous and a few moist râles were audible at the bases. Repeated urinalyses were negative. The hemoglobin was 69 per cent (Dare), the erythrocytes numbered 4,560,000, and the leucocytes, 8,800 for each cubic millimeter. Roentgen-ray studies of the chest verified the fact of cardiac enlargement.*

The diagnosis made was high grade heart-block with convulsive syncope, essential hypertension, and generalized arteriosclerosis. The patient died during a seizure of convulsive syncope.

PATHOLOGICAL EXAMINATION

Necropsy.—The viscera of the chest and abdomen were examined. Since the heart was the organ of interest, the other data will be merely summarized. The other organs appeared grossly and microscopically normal except for the presence of a chronic cholecystitis and cholelithiasis, marked arteriosclerosis of the aorta and large arteries, bilateral hydrocele, and the site of an ancient right inguinal herniotomy with local healed fibrous peritonitis.

The heart weighed 388 gm. On the posterior wall of the right ventricle were two small "soldier's patches," and, on the anterior surface, was a larger, irregular patch measuring roughly 5.0 by 1.5 cm. The epicardial fat was normal in amount. The muscle was grayish-brown, firm, and on tangential section did not show abnormal streaking. The chambers were not dilated and the walls were not hypertrophied. The valves were functionally normal, and the endocardium in general appeared normal. The foramen ovale was closed. Beneath the attachment of the posterior cusp of the mitral valve was felt a number of calcareous deposits, not continuous with one another and not of large size. Bulging out beneath the point of insertion of the anterior leaflet of the mitral valve in the membranous septum was an elongated, irregular cauliflower-like calcareous mass just at the juncture of the membranous and muscular portions of the interventricular septum and embedded in the muscle of the septum. The part which was visible and projecting was about 0.9 cm. in diameter. When the interventricular septum was palpated along the juncture of the membranous and muscular portions, this visible calcareous mass was felt to be continuous with a rigid deposit of calcium of smaller diameter than the projecting mass. This rigid deposit ran toward the right just below the juncture of the membranous with the muscular portion of the septum, becoming gradually smaller, and ending just beneath the middle of the attachment of the right coronary cusp of the aortic valve. On the right side the calcareous mass was felt to project out into the right ventricle prominently beneath the point of juncture of the septal and anterior leaflets of the tricuspid valve, but it was covered by the endocardium and not visible. Fig. 1 is a full-size roentgenogram of the opened heart showing the left side of the interventricular septum with

*The electrocardiographic studies are recorded elsewhere.

the calcium deposit just described. The line of attachment of the aortic cusps is outlined with ink. The visible mass of calcium at the insertion of the anterior cusp of the mitral valve is outlined in ink, while the calcium embedded in the upper end of the muscular portion of the septum is not outlined and is seen extending across to the middle of the attachment of the right coronary cusp of the aortic valve. On the right side of the picture is seen an irregular opacity which is due to the calcium lying beneath the insertion of the posterior leaflet of the mitral valve.

The mitral valve appeared normal except for its association with these calcium deposits. The corpus arantii of the noncoronary cusp of the aortic valve was elongated laterally and thickened, and gave rise to a number of small fibrous processes. The tricuspid and pulmonary valves appeared normal. There was mild atheromatosis of the root of the aorta with one small subintimal calcified plaque,



Fig. 1.—Roentgenogram (full size) of the opened heart through the interventricular septum from the left side, showing the mass of calcium. The portion of the mass which is visible to the unaided eye is outlined with ink. The aortic cusps are also outlined with ink.

0.4 cm. in diameter. The orifices of the right and left coronary arteries appeared normal, but beside each was a small orifice of an independent branch of each artery. There was moderate atheromatosis of both main coronary arteries without notable reduction of the size of their lumina; the smaller branches, however, seemed to be constricted.

Microscopical Examination of the Heart.—Twelve blocks of tissue were removed from the heart for microscopical examination, as follows: (1) one block including the sino-auricular node, which lies beneath the epicardium, in the sulcus terminalis, just below the superior vena cava; (2) three blocks through the auriculo-ventricular and interventricular septums to be described later, which include the main portions of the conduction system; (3) two blocks transversely across the interventricular septum on the left side near the base, which include the main ramifications of the

left bundle-branch; (4) one block from the trabeculum which corresponds to the moderator band of the calf's heart and which normally contains the single right bundle-branch, and (5) one block each from the base of the right anterior papillary muscle, the interauricular septum just below the fossa ovalis, the left ventricle at the base posteriorly, and the posterior wall of the right ventricle midway between base and apex. A block was removed also from the left ventricle, to be stained for fat.

Transverse sections, 10 microns thick, were made at frequent levels through the block which contained the sino-auricular node and were stained with hematoxylin and eosin and with van Gieson's stain. The endocardium and epicardium were not thickened and did not show evidence of inflammation. The sino-auricular node with its special artery appeared normal in respect to the amount of fibrous tissue and the details of the specialized interlacing muscle cells. The arterioles of the node

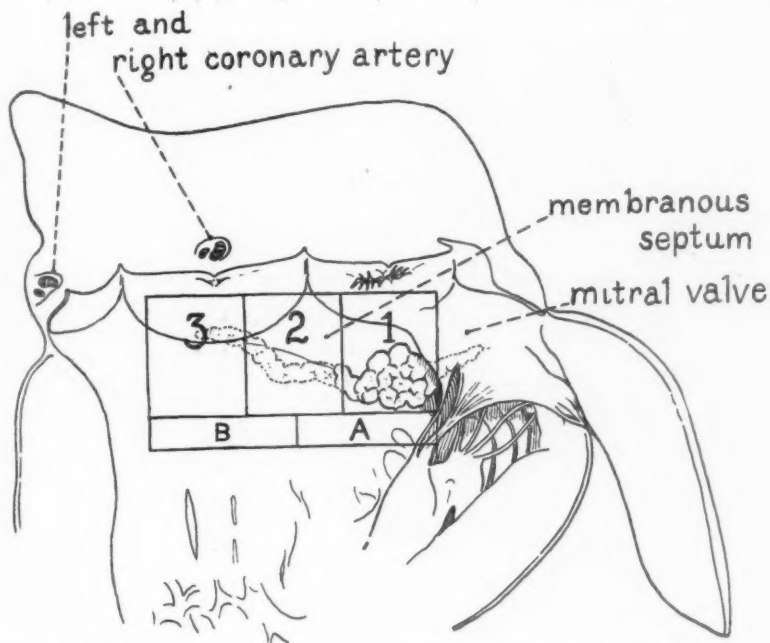


Fig. 2.—Diagram of the left side of the interventricular section, showing the mass of calcium (invisible portion in dotted line) and the site of the blocks of tissue 1, 2, and 3 and A and B.

were not thickened. There were a number of nerves in the vicinity of the node, two of which seemed to be entering the node. The ordinary heart muscle fibers appeared normal, although somewhat distorted as usual, due to the shrinkage during fixation of the large amount of subepicardial fat in this region. The myofibrillae stood out prominently and the cross striations were indistinct and interrupted, but this is the usual appearance of the muscle fibers in the right auricle where fat is excessive. The walls of the blood vessels were not thickened.

The auriculo-ventricular node and bundle were included in Blocks 1 and 2, as indicated in the diagram (Fig. 2) which shows the upper part of the left side of the interventricular septum and root of the aorta. Block 3 contained the main part of the left bundle-branch. Serial sections were made transversely through these three blocks from right to left as one looks at the diagram. Block 1 con-

tained part of the auricular and of the auriculo-ventricular septums and in it were the auriculo-ventricular node (node of Tawara) and the beginning of the bundle of His. Block 2 included part of the membranous and part of the muscular portions of the interventricular septum in the middle area; this contained the main part of the auriculo-ventricular bundle and the origin of both main branches. Block 3 included part of the muscular portion of the septum and the middle third of the right coronary cusp of the aortic valve; it contained part of the left main branch of the bundle. The blocks were decalcified and embedded in paraffin. The sections were cut 10 microns thick, and every twenty-sixth, twenty-seventh, and twenty-eighth section was mounted and stained with hematoxylin and eosin; the twenty-ninth, thirtieth, and thirty-first sections were mounted and stained with van Gieson's preparation. The intervening sections were retained but not mounted or stained except as desired.

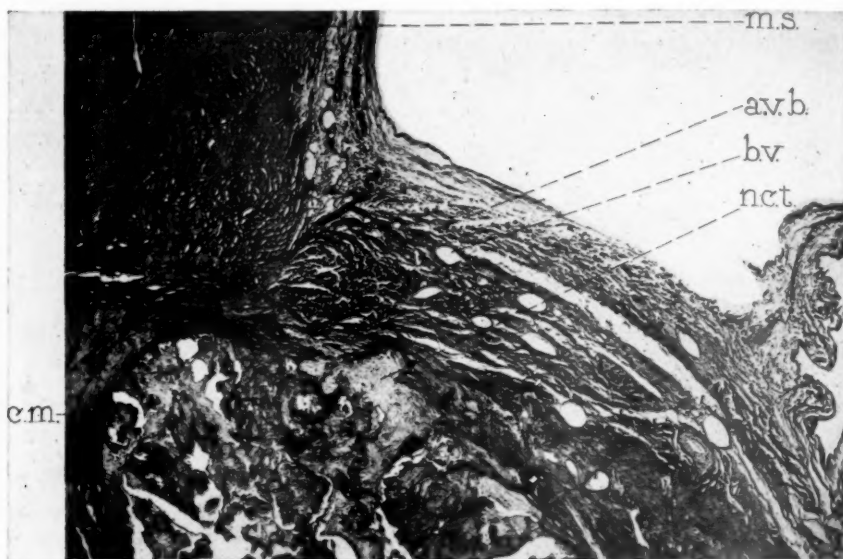


Fig. 3.—Oblique section of the auriculo-ventricular (H's) bundle between the auriculo-ventricular node and the bundle-branches, showing the mass of calcium pressing against it from below (x22); *m.s.*, membranous septum; *a.v.b.*, auriculo-ventricular bundle; *b.v.*, blood vessels in the bundle; *n.c.t.*, new connective tissue between the bundle and the mass of calcium; *c.m.*, calcium mass.

In all of the sections of Blocks 1 and 2 the calcium was present, gradually thinning out in Block 3. The heaviest deposit was in Block 1. It was about 0.6 cm. in diameter at each end, but in the middle it was more extensive because of the portion which protruded into the left ventricle. In the sections, the calcium in Block 1 was seen to be deposited mainly in the ventricular muscle, between the right auricle, above, and the ventricular septum, below, causing the wall of the septum to bulge out on both sides. It was prominent at the base of the aortic attachment of the mitral valve in the substance of which also was seen a little deposit of calcium in one area. The accumulation of calcium was irregular in outline on cross-section and was lobulated; in the main it was enclosed in a capsule of variable thickness composed of dense, collagenous, fibrous tissue. Trabeculums of this capsule were seen in places between lobules of the calcium. The muscular tissue had been pushed aside and at the edges of the deposit of calcium it appeared to be compressed. In some places, narrow strands of muscle fibers formed

a band around the edge of the calcium and were surrounded by or embedded in the fibrous capsule. Between the lobules of the calcium, here and there near the edge of the mass, was a patch of delicate fibrous connective tissue in which were a few small lymphocytes and some plasma cells; these cells were more numerous in some areas than in others. In Block 3 the deposit extended across the interventricular septum at the juncture of the membranous and muscular portions and caused a large hump on the right side in the region of the juncture of the septal and anterior leaflets of the tricuspid valve. The deposit continued in Block 3 until it reached the attachment of the right coronary cusp of the aortic valve, where it thinned out and was lost just above the point of insertion of the cusp in the aortic ring. At the juncture of Blocks 2 and 3 the calcium deposit was about 0.4 cm. in diameter.

In Block 1 just at the base of the mitral valve was a large area of delicate connective tissue with many thin-walled vessels and some small lymphocytes and plasma cells. Where the calcium deposit was heaviest at the base of the valve,



Fig. 4.—Section showing the first part of the right bundle-branch cut almost longitudinally (x36); *t.v.*, tricuspid valve; *f.r.b.b.*, fibrosed right bundle-branch; *n.c.t.*, new connective tissue with plasma cells and small lymphocytes between the bundle-branch and the mass of calcium; *c.m.*, calcium mass.

this connective tissue became more compact and contained smooth muscle fibers from the endocardium and more numerous small lymphocytes and plasma cells. In the middle of this block the central fibrous body began to appear, below and on the left side of which the calcium abutted with a little of the ventricular musculature interposed. No part of the conduction system was recognized until the five hundred twenty-eighth section of Block 1 was reached, where the right edge of the auriculo-ventricular node was seen; nevertheless, the branch of the right coronary artery which was crossing the lower part of the right auricle to supply the node had been seen in the previous sections. The only abnormal artery was noted in the region of the right end of the node. This was a small artery with a very thick muscular wall and a small lumen, situated at the right side of the central fibrous body and later turning downward and running between the central fibrous body and the node. The node was seen in the sections from the five hundred twenty-eighth to the eight hundred seventy-fourth. It was apparently normal and was formed as

usual by compact whorl-like masses of small Purkinje fibers with a delicate interstitial fibrous connective tissue. One large artery and a number of smaller ones were in the node. In the region of the eight hundred seventy-fourth section the node passed into the bundle of His and the fibers became more nearly parallel. The calcium now came to lie more below than on the left side of the bundle, but the latter was for a distance still on the right side of the central fibrous body. In the first sections of Block 2, the bundle was longer transversely than from above downward (Fig. 3). It contained several small blood vessels cut transversely. Below the bundle, and nearer the left side, the calcium was separated from the bundle by very little fibrous tissue and a bit of the calcium projected slightly upward into the bundle. Just to the right of the projection was an area of delicate connective tissue containing a number of thin-walled blood vessels and some plasma cells and small lymphocytes. The deposit of calcium was now pushing out the membranous septum toward the left. A little farther along the area of delicate connective tissue became larger and was interposed between the calcium and the



Fig. 5.—Cross-section through upper portion of interventricular section showing the end of the crus communis and the left bundle-branch in relation to the mass of calcium (x12). *ms.*, membranous septum; *avb.*, bundle of His, considerably fibrotic; *lbb.*, left bundle-branch, the upper portion of which is invaded and almost replaced by fibrous connective tissue; *cm.*, calcium mass; *miv.s.*, muscular portion of interventricular septum.

bundle. The small lymphocytes were at first more numerous. Soon, however, the plasma cells became more abundant and the small lymphocytes scarce. The Purkinje fibers of the bundle were considerably shrunk and distorted due to decalcification and fixing, but otherwise appeared normal. Between the sixty-third and four hundred third sections of Block 2, the bundle expanded toward the right and gave origin to the right main branch (Fig. 4). The branch itself, however, was traceable only as far as the right side of the hump formed in the wall of the right ventricle by the mass of calcium where its pathway seemed to be obstructed by dense fibrous tissue. Between the two sections mentioned, the main bundle became progressively flatter, as though compressed from below by the calcium mass. Also it became more fibrous and was replaced, at its left side, first by fibrous tissue and then by a nodule of calcium. In sections near the three hundred sixty-first the bundle was very flat, very fibrous and almost obliterated. From the four hundred fourth section on, the bundle passed through the membranous septum to the left side, forming

at first the triangular crus communis as it lay in the base of the membranous septum. The crus communis was separated from the subjacent ventricular muscle by the calcium; it was very fibrous and contained a large number of round spaces, evidently blood channels. The fibrosis was most marked at its base adjacent to the calcium mass. The bundle became gradually larger, but this increase in size was due to a greater fibrous content, whereas the muscle elements became scantier. The origin of the left main branch was soon seen and could be traced through several hundred sections. The bundle and the beginning of the left main branch came to be invaded by a few small lymphocytes. By the time the six hundred fifty-second section was reached, the bundle had become replaced almost entirely by fibrous tissue and the origin of the left branch passed through a large area of fairly delicate connective tissue which contained many relatively large blood spaces and some small lymphocytes (Figs. 5 and 6). From this point on, the left branch was seen lower down, to the left of and below the mass of calcium until it was



Fig. 6.—Greater magnification (x33) of portion of preceding section showing the fibrotic bundle of His, *f.a.v.b.*; the large area of delicate connective tissue through which the left bundle-branch, *f.l.b.b.*, passes; and the calcium mass, *c.m.*

no longer present in the sections beyond the four hundred ninety-sixth of Block 3. The left branch itself appeared normal. The deposit of calcium in Block 3 was surrounded by a thicker capsule of dense fibrous tissue, which extended downward for a distance into the ventricular muscle.

Of the two blocks of tissue removed from the left side of the interventricular septum near the base and cut transversely, that nearer the right (Block B, Fig. 2) showed the fibers of the left bundle-branch at the point where they were spreading out. In these sections the Purkinje fibers were seen both in transverse and longitudinal section, directly beneath the endocardium, and appeared normal.

The transverse sections of the moderator band failed to reveal any recognizable elements of the Purkinje system, that is, the right bundle-branch. But in the transverse sections of the base of the right anterior papillary muscle, two bundles of fibers resembling Purkinje fibers were seen, one on either side, embedded in a large amount of fatty connective tissue. The fibers themselves appeared normal but the large amount of fatty connective tissue was distinctly abnormal.

The sections of the interauricular septum and the right and left ventricles showed the cardiac muscle fibers to contain considerable lipochromatic pigment at the nuclear poles, more marked in the ventricles than in the auricles. There was no increase in the interstitial connective tissue and there were no areas of fibrosis. The walls of the arterioles were not appreciably thickened nor were the lumina narrowed. The section stained with Sudan III showed a slight deposition of small fat droplets in the muscle fibers.

Summary of Pathological Examination.—There was a bar of calcium extending across the interventricular septum, at the juncture of the membranous and muscular portions, and embedded in the upper edge of the ventricular musculature. A large portion of the bundle of His was invaded by fibrous tissue, some plasma cells and small lymphocytes, and was almost obliterated in part. The origin of the right bundle-branch was quite fibrotic. The origin of the left bundle-branch passed through a large area of relatively young fibrous connective tissue. The conduction system apparently was not entirely interrupted at any point but its function was certainly markedly impaired by the invasion of fibrous tissue and probably also by compression from the mass of calcium. The cardiac muscle fibers contained polar lipochromatic material but there was no fibrosis, very little fatty degeneration was present and the arterioles were not appreciably thickened.

Comment.—The frequency and the severity of the seizures of convulsive syncope were prominent features of this case. In several of the attacks, death appeared to have occurred but temporary recovery ensued. From the time that death appeared imminent until death actually occurred, a period of fifty hours elapsed.

During one of the most marked seizures, complete cardiac asystole occurred for approximately four minutes. This seems almost unbelievable but is proved by the continuous electrocardiographic records obtained. A somewhat comparable example of prolonged asystole is found in the case reported by Levine and Matton in which ventricular fibrillation and asystole occurred for five minutes.

Ventricular asystole occurred at times associated with high grade block, on one occasion to a degree of 64:1 and again in association with complete cardiac asystole.

The correlation in this case of the clinical features and the pathological observations is of considerable interest.

DISCUSSION OF ELECTROCARDIOGRAMS

The electrocardiograms in this case revealed multiple transitions which ranged from periods of normal sinus rhythm through varying grades of block, to remarkably long periods of complete cardiac asystole.

At the time of the admission of the patient to the hospital, 3:1 partial heart-block was present (Fig. 7). The ventricular components in Leads I and II are normal in appearance, but in Lead III the QRS complexes are of very low amplitude and are notched.

On the following day (Fig. 8) 2:1 partial block was detected. The similarity of the general features between this electrocardiogram and the one obtained on the preceding day is apparent.

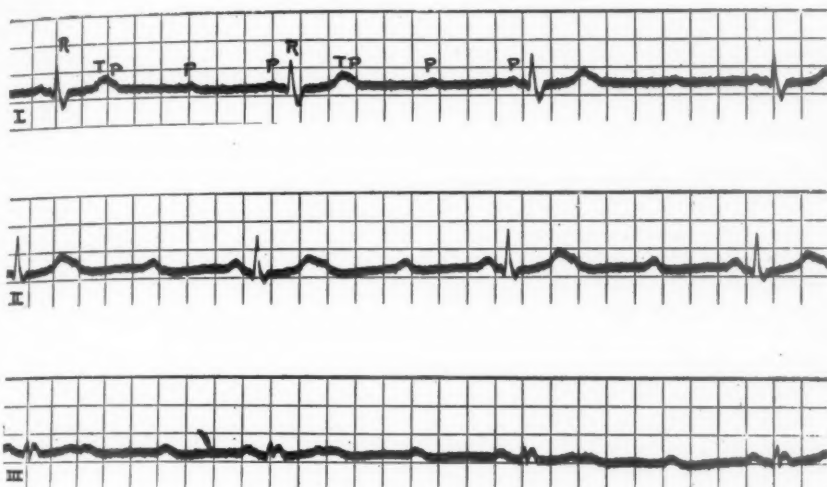


Fig. 7.—This record, taken in all leads, was obtained on the day of the patient's admission to the hospital. It shows the presence of 3:1 partial block.*

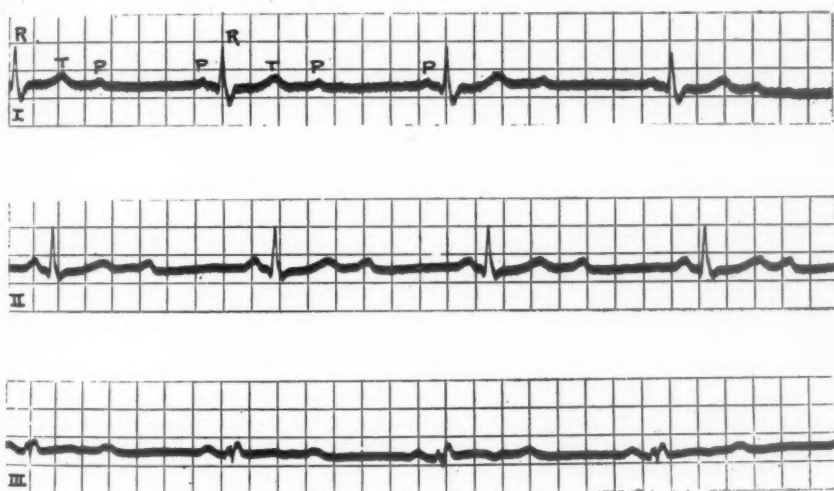


Fig. 8.—This record, taken in all leads, was obtained the following day (Fig. 7) and shows 2:1 partial block.

The records obtained two days later (Figs. 9 and 10) show the re-establishment of normal sinus rhythm, occasionally interrupted by periods of block. These events are of interest in view of the pathological

*In all the records, the horizontal lines represent 1 millivolt and the vertical lines 0.2 second.

data; even though the auriculo-ventricular bundle was extensively involved, it was still able to conduct impulses in a normal manner for short intervals.

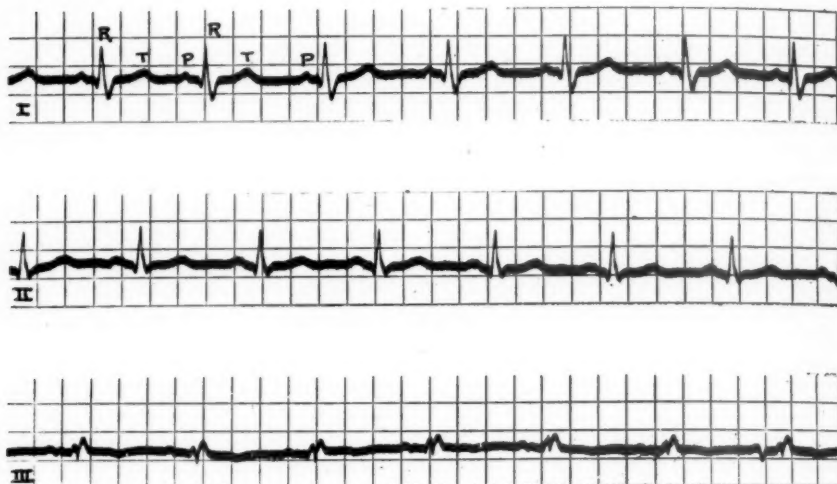


Fig. 9.—This record, also taken in all leads, was obtained two days later (Fig. 8) and shows the temporary restoration of normal sinus rhythm.



Fig. 10.—This record was obtained the same day (Fig. 9) and portrays sinus rhythm with periods of block.

The succeeding records (Figs. 11 to 26) were obtained about a week later, during the time when the seizures of convulsive syncope were extremely numerous. They were all taken in Lead II and are continuous strips.

All degrees of block are shown. In Figs. 11 and 12 are seen records of complete heart-block with varying ventricular complexes, indicative probably of changes in the point of origin of the idioventricular rhythm.

In Fig. 12 is shown the onset of a long period of ventricular asystole with high grade block, 22:1. In one electrocardiogram, a period of 64:1 block occurred. The diminution in the amplitude of the P-waves and the associated arrhythmia are shown in Fig. 14.

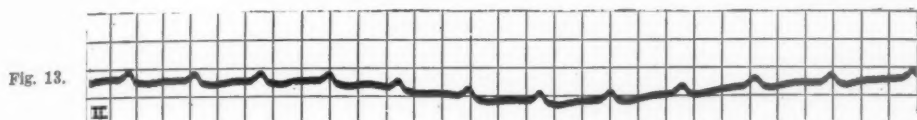
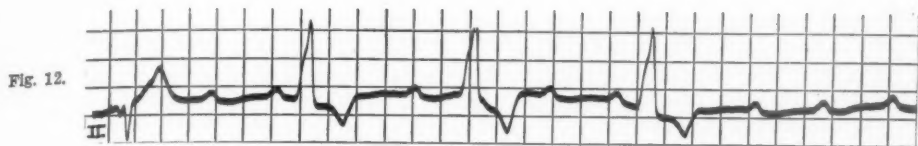


Fig. 11.—This record, and the following sixteen records, obtained about a week later than this one, are continuous strips taken in Lead II. They show many transitions, with some rather unusual features. In this record is shown complete heart-block with temporary aberration of the initial ventricular complexes.

Fig. 12.—This record is similar to the preceding one and shows the onset of a prolonged period of ventricular asystole.

Fig. 13.—This record exhibits a series of auricular waves only.



Fig. 14.

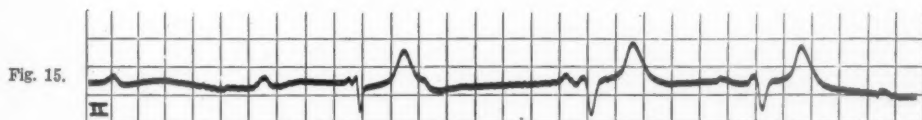


Fig. 15.

Fig. 14.—This record likewise shows only auricular complexes. Note the variation in the amplitude of the P-waves and the evident arrhythmia.

Fig. 15.—This record shows ventricular recovery. Note the change in direction of the ventricular complexes presumably indicating a shifting origin of the idioventricular rhythm.

The resumption of ventricular activity is shown in Fig. 15. The ventricular components are completely reversed but recovery took place.

Figs. 16, 17 and 18 also show a period of high grade block with recovery.

Remarkably long periods of complete cardiac asystole are recorded in Figs. 19 to 24, and long periods of ventricular asystole with high grade block, in Figs. 25 and 26.

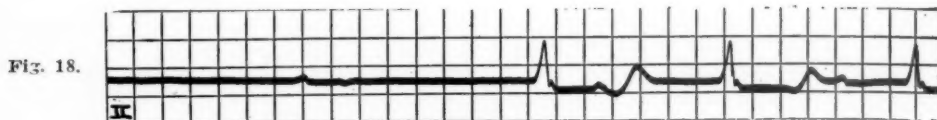
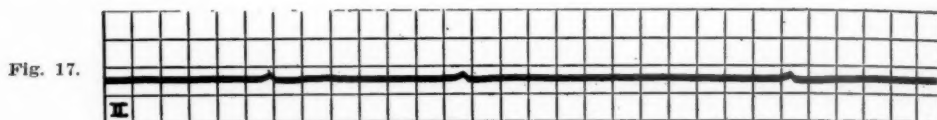
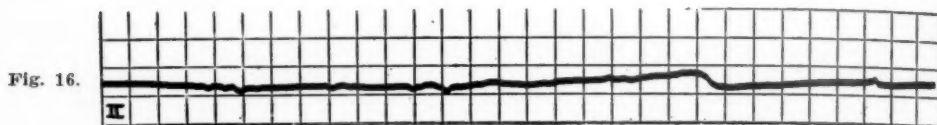


Fig. 16.—This record shows a long period of ventricular asystole with only occasional P-waves. Some of the first irregular undulations are probably artefacts.

Fig. 17.—This record shows continued ventricular asystole with only three auricular waves, occurring irregularly.

Fig. 18.—This record shows ventricular recovery.



Fig. 19.—This record displays a remarkably long period of complete cardiac asystole.

Fig. 20.—This record is identical in character with the preceding one but successively continuous in time.

Fig. 21.—This record shows the continuation of complete cardiac asystole.

The high grade block exhibited at times in this case is not unlike that seen in the dying human heart. Under conditions of impending death,

when evidence of disease of the conduction paths has not been apparent, the appearance of varying degrees of block has been attributed to asphyxia. It is not unlikely, therefore, that some of the phenomena observed in this case were, in part at least, influenced by asphyxial states.

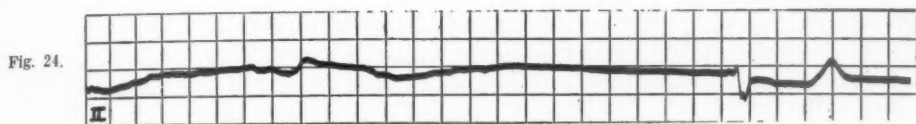
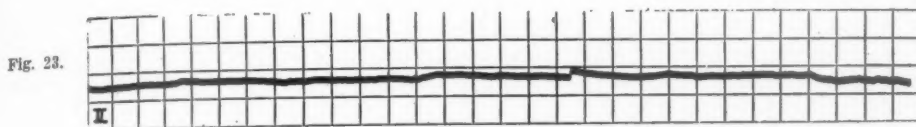
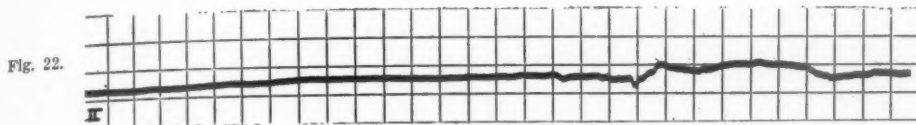


Fig. 22.—This record shows complete cardiac asystole and one peculiar deflection which may represent an attempt at ventricular activity.

Fig. 23.—This record shows complete cardiac asystole. The slight deflection shown appears to be an artefact.

Fig. 24.—This record shows temporary ventricular activity.

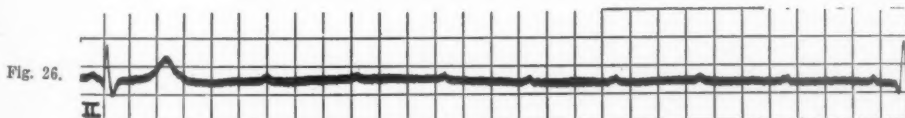


Fig. 25.—This record shows 10:1 block.

Fig. 26.—This record likewise shows high grade heart-block.

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THE DISTORTION OF THE ELECTROCARDIOGRAM
BY ARTEFACTS*

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ARTEFACTS of various kinds are encountered in varying degrees in any large series of electrocardiograms, their number depending in part on the accuracy of the technic employed and in part on the types of individuals electrocardiographed. To those of wider experience such artefacts are usually readily recognizable, but to those whose experience has been more limited, they are a frequent source of perplexity in the interpretation of the tracings. In a rather large percentage of the electrocardiograms that have been referred to this laboratory for an opinion, artefacts have been found responsible for the difficulties that have been met in their analysis. For this reason we believe that a brief account of our experiences with electrocardiographic artefacts may prove helpful. This experience we are summarizing in this paper.

The literature makes little mention of the distortion of the electrocardiogram by artefacts, although certain writers, in particular S. Calvin Smith,[†] have illustrated a number of them. Texts on electrocardiography, as a whole, devote very little space to the subject.

We have examined the 15,600 electrocardiograms[‡] that have been taken in this laboratory during the past several years, and have selected those showing artefacts. These we have divided into several groups, depending upon their cause, and have chosen one of the most typical from each of the more important groups or subgroups for illustration.

Artefacts are usually about evenly divided between those of intrinsic origin, that is arising within the individual himself and those of extrinsic origin, that is, arising from instrumental factors outside of the body. These intrinsic and extrinsic factors are in turn divided into subgroups.

*From the Cardiographic Laboratory of the Massachusetts General Hospital, Boston, Mass.

[†]Smith, S. Calvin: *Heart Records, Their Interpretation and Preparation*, 1923, F. A. Davis Co.

[‡]These electrocardiograms were taken with the string galvanometer manufactured by the Cambridge Scientific Instrument Company of Cambridge, England, in 1914. The observations in this paper refer to the string type of galvanometer but are also applicable in part to other types of galvanometers.

A. INTRINSIC FACTORS

The intrinsic factors are in the main due to somatic muscular activity.

1. *Muscular Contractions*.—When there is a single contraction or movement of a large skeletal muscle or group of such muscles, there is often a considerable effect produced upon the string of the galvanometer. Depending upon the movement various effects are recorded, from the raising of the base line to simulate a P- or T-wave or a QRS complex to the forcing of the string beyond the limits of the photographic field. In Figs. 1, 2, and 3 this condition is illustrated: as a single muscle movement in Fig. 1, as a recurrent facial, arm and

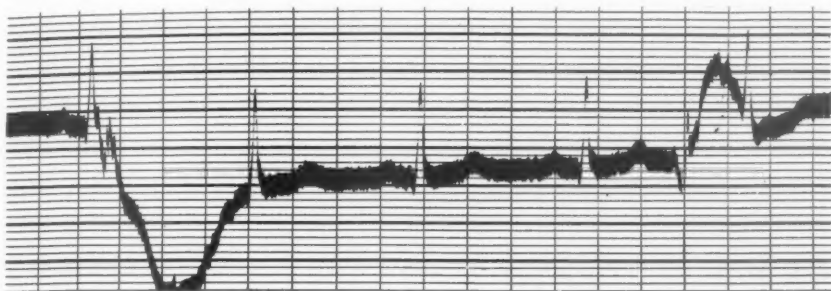


Fig. 1.—Artefacts in electrocardiogram (Lead I), consisting in a displacement of the base line due to muscle movement (contraction and movement of left hand and later of the right hand in electrodes). Times lines represent fifths of a second, amplitude is expressed in 10^{-1} volts in this and in the following figures.

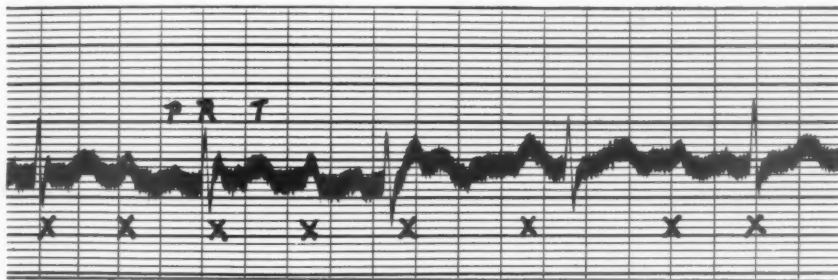


Fig. 2.—Electrocardiographic artefacts (Lead II) due to a regularly recurring tic involving the arms and head. The deflections due to the tic are marked by an x.

shoulder tie in Fig. 2, and as the constant rapid distortion of paralysis agitans in Fig. 3. Strange as it may seem, these artefacts in unskilled hands are often the most difficult to interpret, since the individuals electrocardiographed have not been closely observed at the time for the detection of such abnormal muscle action, and unusual P-, QRS, or T-waves have been thought present. The larger the muscle and the nearer to the electrodes, the greater is the distortion that results from its contraction.

2. *Somatic Tremor*.—Undoubtedly the most frequent type of all artefacts is due to varying degrees of somatic or nervous tremor (Fig. 4).

This is the result of the fine rapid action of fibers in the somatic musculature from the tension of nervousness or hyperthyroidism or actual spastic contraction. It is at times present to an extent sufficient to mask the cardiac deflections.

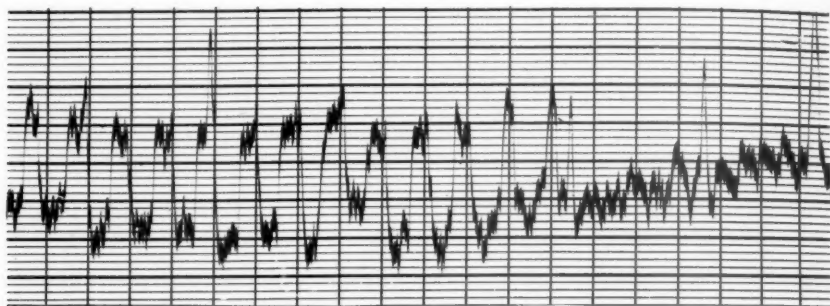


Fig. 3.—Artefacts due to paralysis agitans. The coarse skeletal muscular movements occurring about five times a second caused striking regular wide deflections of the base line, at times burying the auricular and ventricular deflections, but at other times the QRS group can be easily made out superimposed on the artefacts. At the end of the record auricular flutter is simulated.

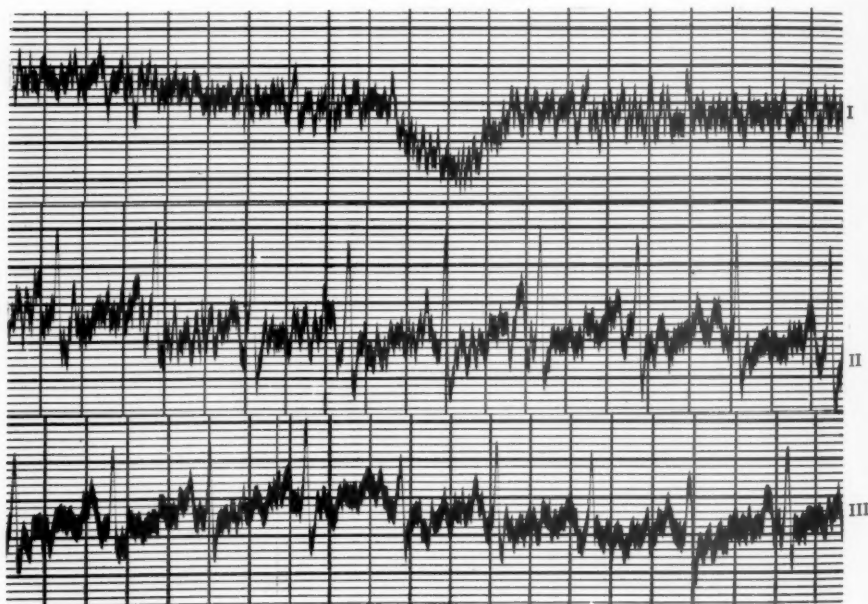


Fig. 4.—Electrocardiogram showing marked somatic or "nervous" tremor deforming all three leads. In Lead I it is particularly difficult to distinguish any deflections of cardiac origin.

3. *High Resistance With Loose String.*—In some cases in which the so-called skin resistance is high, the string of the galvanometer must be loosened excessively to allow proper standardization, a deflection of one centimeter for one millivolt. The degree of loosening may result in a marked deformation of the deflections, and sometimes in such

cases an overshooting of the string also occurs. This condition is not uncommon and an example is shown in Fig. 5. If marked, the deformity of QRS or T-waves may simulate some important pathological conditions, like intraventricular block or the abnormal T-wave of coronary disease. It should also be observed that high resistance (over 2000 ohms) frequently originates not in the individual, but in

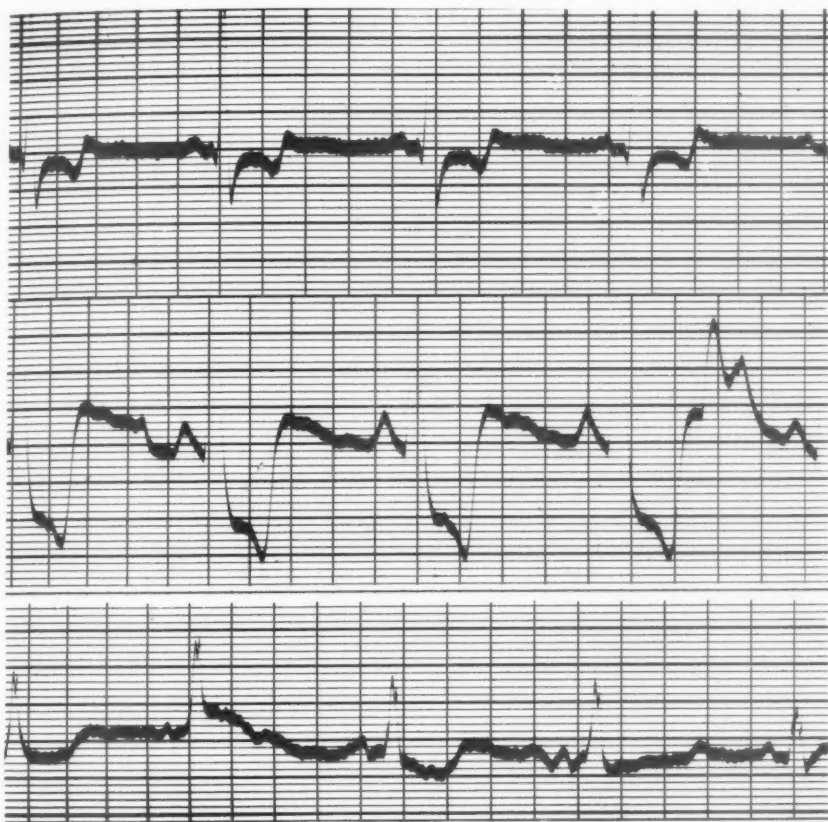


Fig. 5.—Electrocardiogram (Leads I, II, and III) showing artefacts due to high resistance and polarization. Also at the end of Lead II there is an artefact due to somatic muscle movement.

the instrumental connections, if for example the electrodes themselves are faulty, as when fresh water instead of salt water is used or when rust collects on the terminals, especially in humid weather.

B. EXTRINSIC FACTORS

1. *Polarization of Electrodes.*—As Einthoven, Lewis, Pardee, and others have pointed out, polarization of electrodes may cause important artefacts, chiefly shown by overshooting of the string and a movement back to or beyond the base line even when a constant current

is maintained. This neutralizing or exaggerating effect of polarity is largely avoided by the use of nonpolarizable electrodes, but for practical purposes polarizable electrodes may be employed, with only oc-

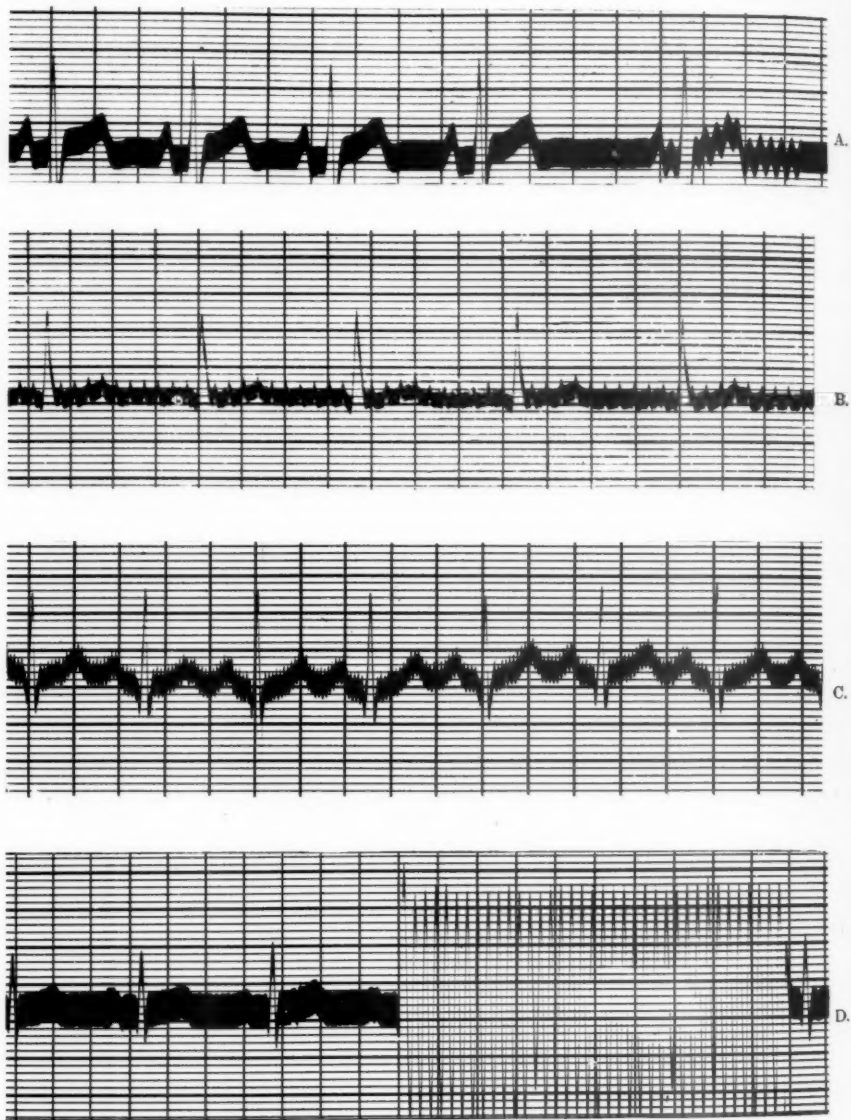


Fig. 6.—Electrocardiograms (A, B, C, and D) illustrating various rhythmic disturbances of the base line due to induction from outside currents at varying rates and strength. A and D represent short intervals of artefact due to the ringing of a telephone bell. B and C represent constantly recurring distortions, at a cycle of 15 per second in B and of 60 per second in C.

casional distortion of the electrocardiogram, provided the surface contacts between patient and electrodes are large. Nevertheless it is important always to be on guard against the artefacts due to polariza-

tion in using polarizable electrodes. Fig. 5 illustrates this artefact as well as that resulting from too high a resistance.

In addition to the high resistance and polarization of the electrodes there are a number of other important extrinsic causes of artefacts.

2. *Outside Current (Induction).*—In Fig. 6 is illustrated a type of artefact occurring much more frequently in the past than at present when electrocardiographic equipments are better constructed and protected. It is produced by the effect on the string of an outside current, for instance from a ringing telephone or an adjacent x-ray machine. A series of regular waves is produced, at times so marked as to be easily recognized, but in some instances more confusing because only the base line may appear to be affected. Four types of varying rate and amplitude are illustrated in Fig. 6.

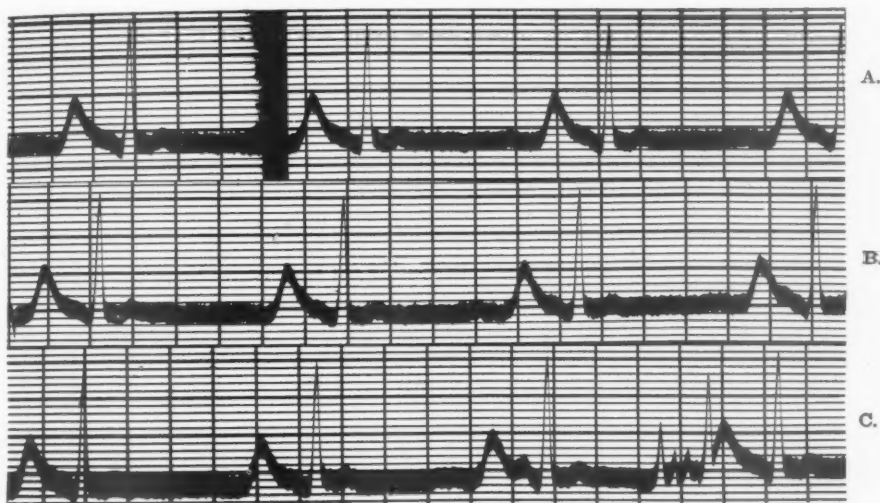


Fig. 7.—Artefact in electrocardiogram due to faulty contact shown near end of third strip (C). The plate was wrongly placed in the camera with the film side away from the light resulting in reversion of record when printed in routine way. The second thus reads from right to left. Also a black band is seen in A, due to flickering of the arc light.

3. *Faulty Contacts.*—Another type of artefact is that caused by a switch defect, commonly in the Wheatstone bridge of the electrocardiograph. Loose or dirty contacts are usually responsible. In this case the tracing shows a series of uneven and irregular lines interrupting the cardiac deflections for usually not more than a few fifths of a second. An example of this is shown in Fig. 7. This illustration also shows faulty technic in photography. The plate was reversed end for end with the result that all leads read from right to left. We have known instances of the incorrect interpretation of the P-wave as the T and the T-wave as the P when this reversion has occurred.

4. *Reversed Electrodes.*—If the lead wires are not properly applied, a confusing picture may be produced.* Thus in Fig. 8, in which this error has been made, it will be seen that a reversion of the leads explains the difficulty. The wires attached to the terminals of the right arm and left arm have been reversed, resulting in Lead I being recorded upside down, Lead III being recorded in the place of Lead II, and Lead II recorded in the place of Lead III.

Lead

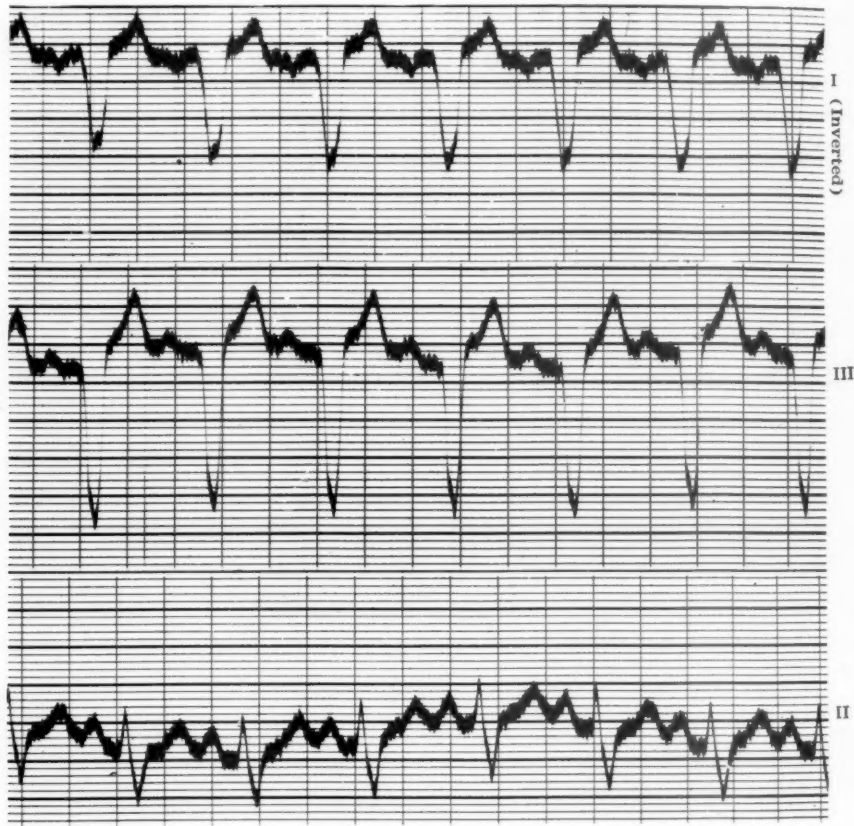


Fig. 8.—Artefact in electrocardiogram due to crossing of leads. The right and left arm wires were wrongly attached (reversed), resulting in inversion of Lead I and transposition of Leads II and III. Right bundle-branch block is present, but at first glance in the uncorrected record the interpretation appears confusing. The inversion of P in Lead I helps to reveal the error.

5. *Composite Electrocardiograms.*—Fig. 9 illustrates a double electrocardiogram. The leads were applied to a child who was held by the mother in her lap during the taking of the tracing. As will be seen the mother's heartbeat is recorded along with that of the child.

In addition to these artefacts there may occur a few others. At times at the beginning of a tracing the time marking wheel, if such is

*Error in the wiring of wards by electricians may result in this same artefact even though the electrodes themselves appear properly applied.

used, may be started at too rapid a rate and may continue to revolve at double speed throughout the tracing. Thus the time lines will separate tenths of a second instead of the customary fifths. Again in those instruments in which plates are used and the speed of the plate is controlled by an oil chamber, if an air bubble gets into the oil when the cylinder is not sufficiently filled, the plate will move faster than its accustomed rate for a short interval, resulting in the widening of the time markings and a distortion of the tracing. A further distortion of the electrocardiogram may come from irregular movement of plate, or roll of paper or film, in the camera due to incorrect working of the mechanism. This, of course, can almost always be easily detected.*

Artefacts due to inexact standardization are, of course, not recognizable in themselves on inspection of the electrocardiogram but comparison of several records from the same patient, perhaps taken on different occasions, will reveal this error. Naturally one assumes that

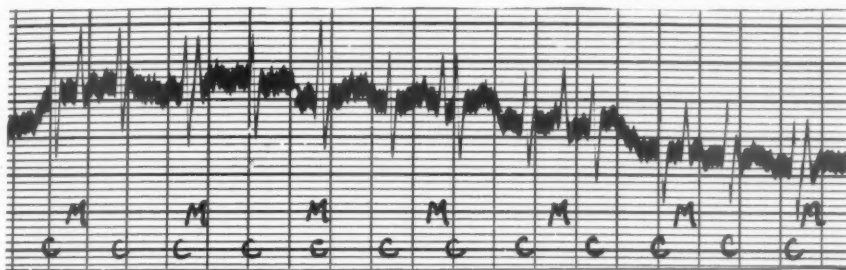


Fig. 9.—Artefact due to composite electrocardiograms of Lead I of infant and of mother who held the baby's arms with her own hands. The child's ventricular complexes are marked *C* and the mother's *M*. The auricular deflections are not evident. The infant's heart rate was 180 per minute and the mother's 100.

this type of artefact is routinely guarded against at the time the electrocardiogram is taken.

Of course there may also arise errors in photography, recording, and filing, but these are simple and quickly recognized and need not concern us further.

SUMMARY

The frequency with which the electrocardiogram may be distorted by artefacts, especially since the widespread use of the galvanometer in clinical work, demands more consideration than is usually accorded it.

We have presented a brief discussion of the more important of these artefacts that have come to our notice in the past thirteen years, and have divided them into two groups, as they may be intrinsic or extrinsic in origin. Illustrations of the more important artefacts have been added.

*If an arc light is used, flickering or sputtering due to carbon impurities or to faulty adjustment may streak the record.

Artefacts of intrinsic nature may arise from single contractions or movements of skeletal muscles, from repeated contractions of such muscles, as in a tic, from paralysis agitans, and finally from the tension of muscles constantly in contraction (called somatic tremor), as in nervous or hyperthyroid individuals. The high resistance of the subject or of the instrument itself may also give rise to artefacts due to the inertia of a loosened string.

Artefacts of extrinsic origin include those due to high resistance, polarization of electrodes, outside current, faulty contacts, reversed leads, composite electrocardiograms, and errors in photography or in the manipulation of the time marker.

HEART MURMURS: THEIR INCIDENCE AND INTERPRETATION

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L AENNEC'S experience with heart murmurs may be said to typify the confusion and uncertainty that have surrounded this subject since the great Frenchman devised the stethoscope and made it possible for us to hear the sounds that are generated over the precordium. It is said that in the beginning he looked upon all of the murmurs that he heard as indicative of disease of the cardiac valves. But after following many of the patients in whom he had heard such murmurs to the autopsy table, and finding no anatomical confirmation of his ante-mortem impressions, he concluded that murmurs over the heart were entirely without significance.¹ We know now that both of his conclusions were wrong; that some of the murmurs are indicative of defects in cardiac structure; that many of them point to no such defects, and that careful examination and discriminating judgment will enable us properly to differentiate them.

In my work as a consulting physician I became impressed with the frequency with which innocent murmurs over the precordium were interpreted as indicative of cardiac pathology, and the patients reduced to the unhappy state of cardiac neurasthenics. This unfortunate state of affairs was not infrequently encountered after routine examinations for life insurance, healthy applicants leaving the examiners' hands with their attention attracted to their hearts for the first time, and with a fear of heart disease fixed upon them. Other patients were seen who had been advised of these murmurs following examinations that had been made in the course of trivial illnesses, and who had had unnecessary physical restrictions placed upon them because of the assumption that the murmurs demanded protection of the heart muscle.

It occurred to me that it might prove interesting and serviceable to collect data concerning the incidence and significance of murmurs heard over the precordium in the course of routine office examinations. With this idea in mind, I paid especial attention to all such murmurs in a series of 1166 consecutive cases.

In doing this work I adopted the following classification of heart murmurs:

I. *Organic Murmurs*.—Under this heading were listed all murmurs that were interpreted as indicative of organic change in the structure

of the heart valves, those engendered by a disease of the aortic arch, and those indicating a persistence of embryonic openings between the chambers of the heart or a persistent ductus botalli.

II. *Accidental Murmurs*.—Under this broad heading were classed all murmurs which were interpreted as being without significance of organic change in the structure of the heart valves, and not dependent upon any form of congenital heart disease or abnormality in the aortic arch. These murmurs were subdivided as follows:

1. Intracardiac accidental murmurs.
2. Extracardiac accidental murmurs.

The intracardiac accidental murmurs were looked upon as those which probably found their origin inside of the heart as the result of a dilatation of the valvular rings (inorganic murmurs), because of a change in the quality of the blood (hemic murmurs), or because of a deformitory of a valvular orifice produced by a displacement of the heart, as in pleural effusion. The extracardiac murmurs were regarded as arising outside of the heart and were clearly demonstrated to be dependent upon some phase of respiration. Under this head one murmur was designated as pleuropericardial because of its superficial, scratchy character and its persistence in all phases of respiration.

The criteria for the grouping of the murmurs were as follows:

1. A careful history, with emphasis on the past or present occurrence of cyanosis, dyspnea and edema, the rheumatic triad, and syphilis.
2. The time and quality of the murmur and its transmission, if any.
3. The association of the murmur with thrills, accentuation of the second sounds, and the height of the systolic and diastolic arterial pressures.
4. The presence or absence of anemia.
5. The presence or absence of cardiac hypertrophy as determined by percussion and roentgenographic study.

TABLE I

| | | PER CENT |
|--------------------------------|------|----------|
| Total number of cases examined | 1166 | |
| Murmurs heard in | 166 | 14.2 |
| Accidental murmurs | 127 | 10.9 |
| Organic murmurs | 39 | 3.3 |
| Mitral insufficiency | 15 | 1.3 |
| Mitral stenosis | 8 | 0.7 |
| Aortic insufficiency | 10 | 0.8 |
| Dilated aorta (3) Aneurysm (1) | 4 | 0.3 |
| Congenital heart lesion | 2 | 0.2 |

It will be noted from Table I that accidental murmurs were heard in 10.9 per cent of the patients examined. Potain,² the great French cardiologist, reported an incidence of such murmurs in 12.5 per cent of all of the patients seen in his hospital service. He found them with especial frequency in association with exophthalmic goiter, chlorosis, and the acute fevers. Thayer³ reported murmurs of this type in 56.4 per cent of patients in the first decade of life, with a gradually diminishing incidence according to decades up to the fourth where he found murmurs in 19.2 per cent. These figures certainly tend to emphasize the importance of the subject.

TABLE II

| | | PER CENT |
|---------------------------|-----|----------|
| Total number of murmurs | 166 | |
| Accidental murmurs | 127 | 75.6 |
| Mitral insufficiency | 15 | 9.0 |
| Mitral stenosis | 8 | 5.0 |
| (Total mitral murmurs | 23 | 13.9) |
| Aortic insufficiency | 10 | 6.0 |
| Dilated aorta or aneurysm | 4 | 2.3 |
| Congenital heart lesions | 2 | 1.2 |

In Table II we see that 75 per cent of the murmurs heard over the precordium in this series are of no pathological significance. This should serve to emphasize the very great care that should be exercised before a murmur is interpreted as indicative of a cardiac lesion.

TABLE III
SEX INCIDENCE

| | NUMBER | PER CENT |
|---------|--------|----------|
| Males | 46 | 37 |
| Females | 81 | 63 |

In this series accidental heart murmurs were heard almost twice as frequently in females as in males. Of the 1166 patients examined, 549 were males and 617 females. Thus, the indications are that accidental murmurs are to be expected with much greater frequency in the females.

TABLE IV
INCIDENCE OF ACCIDENTAL MURMURS ACCORDING TO DECADES

| DECADE | MURMURS | PER CENT | NO. OF PATIENTS | PER CENT |
|---------|---------|----------|-----------------|----------|
| First | 3 | 2.5 | 27 | 11.0 |
| Second | 11 | 8.6 | 103 | 10.7 |
| Third | 43 | 33.0 | 269 | 16.0 |
| Fourth | 33 | 26.0 | 265 | 12.5 |
| Fifth | 21 | 16.0 | 207 | 10.0 |
| Sixth | 10 | 8.0 | 154 | 6.5 |
| Seventh | 5 | 4.0 | 110 | 4.5 |
| Eighth | 1 | 0.5 | 31 | 3.2 |

The figures in Table IV show both a relative and an absolute maximum incidence of accidental murmurs in the third decade; but it seems to me that the most important showing of all is that these murmurs may be encountered in any decade of life, and that we should be ever on guard to interpret them properly.

TABLE V
SITE AT WHICH ACCIDENTAL MURMURS ARE BEST HEARD

| | NUMBER | PER CENT |
|--------------------------|--------|----------|
| At the base of the heart | 61 | 48 |
| At the apex of the heart | 28 | 23 |
| Audible at apex and base | 99 | 78 |
| Equally at apex and base | 34 | 26 |
| Audible at aortic area | 6 | 4 |
| Total heard at base | 107 | 84 |

Table V serves to stress the importance of the well recognized fact that accidental murmurs are most often heard at the base of the heart. In only 16 per cent of the cases were the accidental murmurs heard at the apex alone. It might be said with propriety that a systolic murmur at the base of the heart should be considered innocent until proved otherwise.

TABLE VI
TIME AND QUALITY OF ACCIDENTAL MURMURS

| | NUMBER | PER CENT |
|-------------|--------|----------|
| Systolic | 127 | 100 |
| Diastolic | 0 | 0.0 |
| Soft | 88 | 70.0 |
| Loud | 35 | 27.0 |
| Musical | 17 | 13.0 |
| Superficial | 10 | 8.0 |
| Scratchy | 1 | 0.8 |

Table VI shows that all of the accidental murmurs were systolic in time. Diastolic murmurs of this type are said to occur,⁴ but I failed to find a single instance in this series. The great majority of these murmurs are classed as soft, but the loud, musical, and superficial ones are sufficiently numerous to keep us from generalizing as to the significance of these qualities. It is well known that the qualities of murmurs that are found in association with organic valvular disease vary tremendously and have little significance, especially as regards loudness and softness, or more properly, the intensity of the murmur, and this seems to apply equally well to the accidental murmurs. I should say that the quality of a murmur is no criterion for decision as to its accidental or pathological generation.

It is well known that in contrast with organic murmurs the accidental ones are not well transmitted in any direction. Of the 12 accidental murmurs in this series that were transmitted toward the axilla

TABLE VII
TRANSMISSION OF ACCIDENTAL MURMURS

| | NUMBER | PER CENT |
|----------------------|--------|----------|
| To axilla from apex | 12 | 9.4 |
| Upward from the base | 1 | 0.8 |

from the apex, 10 of them were of the cardiorespiratory type with the murmur definitely dependent upon a phase of respiration, 1 of them was classed as a hemic blow in association with a hemoglobin of 68 per cent, and 1 as an inorganic murmur in connection with cardiac hypertrophy and dilatation resulting from a long-standing hypertension, the patient having a systolic pressure of 180 mm. and a diastolic pressure of 120 mm. The murmur which was transmitted upward from the base was regarded as an inorganic one due to relative narrowing of the aortic ring in association with a pronounced hypertension.

The pulmonic second sound was found to be accentuated in 5 instances, or 4 per cent of the cases, in which accidental murmurs were heard. Three of these patients showed relatively insufficient myocardial function in connection with hypertension, adiposity, or both, and one was found in association with a pleural effusion. In all of these cases sufficient cause was found to account for an increased pressure in the pulmonary circulation without the existence of an organic valvular insufficiency. In one case no explanation could be offered for the accentuated second sound. These figures serve to emphasize the importance of an unaccentuated second pulmonic sound in establishing the innocence of precordial systolic murmurs.

TABLE VIII
ENLARGEMENT OF HEART AND AORTIC DILATATION IN ASSOCIATION WITH
ACCIDENTAL MURMURS

| | NUMBER | PER CENT |
|---------------------|--------|----------|
| Cardiac hypertrophy | 11 | 8.6 |
| Aortic dilatation | 5 | 4.0 |

In the interpretation of cardiac murmurs it may be accepted as an axiom that an insufficient valve must result in an increase in the size of the heart. The only exception to this rule is in acute valvular disease where the insufficiency of the valve has not lasted long enough for the added demands upon the myocardium to produce hypertrophy of the heart muscle; however, cardiac hypertrophy may be found in association with accidental murmurs as well. This series shows such hypertrophy in 11 instances, or in 8.6 per cent of the murmurs recorded as accidental. In 8 of these cases the hypertrophy was ascribed to pronounced systemic hypertension; in one to a severe ane-

mia;⁴ in 1 to extreme adiposity, and in 1 to a long neglected pleural effusion. Hypertension was regarded as the explanation of all of the instances of aortic dilatation. In other words, an adequate cause, other than an organic disease of the heart valves, was present to account for the enlargement of the heart or the aorta in all the cases so grouped.

TABLE IX
CLASSIFICATION OF ACCIDENTAL MURMURS

| | NUMBER | PER CENT |
|----------------------|--------|----------|
| Intracardiac | 36 | 28.3 |
| Inorganic | 12 | 9.4 |
| Hemic | 21 | 16.0 |
| Cardiac displacement | 3 | 2.3 |
| Extracardiac | 91 | 71.7 |
| Cardiorespiratory | 90 | 70.9 |
| Pleuropericardial | 1 | 0.8 |

Table IX shows the great preponderance of cardiorespiratory murmurs over accidental murmurs of other types. It is fortunate that this type of murmur is the most easily differentiated from murmurs arising from valvular heart disease. These murmurs can be clearly related to the juxtaposition of the heart and lung, disappearing on some phase of respiration, being more clearly heard at the base than at the apex, and tending to diminish in intensity or disappear with the patient in the upright position. The so-called hemic murmurs should likewise give rise to little confusion when their association with more or less pronounced anemia is kept in mind. The inorganic type of accidental murmur requires more careful differential study for its recognition, and its practical importance as an accidental murmur is not so great, as it is truly indicative of cardiac embarrassment and must be looked upon as significant.

TABLE X
RELATIONSHIP OF ACCIDENTAL MURMURS TO BODY WEIGHT

| | NUMBER | PER CENT |
|---------------|--------|----------|
| Underweight | 84 | 66.0 |
| Normal weight | 21 | 16.0 |
| Overweight | 22 | 17.0 |

As would be expected, accidental murmurs are most frequently heard in patients who are below their optimum weight, for the asthenic type of person, with the long and narrow chest, has the left border of the heart and the anterior lappet of the left lung in close apposition. These are optimum conditions for the generation of cardiorespiratory murmurs; but it is important to remember that such murmurs are to be heard also in patients who are at or above their proper weights.

TABLE XI
BLOOD PRESSURE, PULSE AND HEMOGLOBIN

| | NUMBER | PER CENT |
|------------------------|--------|----------|
| Blood pressure | | |
| Below 111 mm. systolic | 45 | 35.0 |
| Above 130 mm. systolic | 30 | 23.0 |
| Normal | 52 | 41.0 |
| Pulse | | |
| 70 to 90 | 65 | 51.0 |
| Below 70 | 9 | 7.0 |
| Above 90 | 53 | 41.0 |
| Hemoglobin | | |
| Below 75 per cent | 41 | 32.0 |
| Above 74 per cent | 86 | 67.0 |

The blood pressure and pulse rate seem to be practically unrelated to the incidence of accidental heart murmurs. The exception to this statement is in the fact that long-standing hypertension brings about changes in the heart and aorta that produce murmurs of the inorganic variety. Accidental murmurs are not heard with any greater frequency in association with hypotension than they are in patients with normal blood pressure. The pulse rate was within normal limits in 51 per cent of the cases. In the presence of pronounced anemia we naturally expect to find "hemic blows" over the precordium, but these statistics show that 67 per cent of the patients with accidental murmurs had no anemia.

In 6 of these patients with accidental heart murmurs, or 4.7 per cent, a diagnosis of organic heart disease had been made on the basis of the murmur alone. In all of these cases the murmurs were of the cardiorespiratory type, and the mistake should have been easily avoided.

CONCLUSIONS

1. In 1166 patients 127 accidental heart murmurs were discovered, while 39, or 3.3 per cent of them, showed murmurs indicative of organic valvular disease.

2. All of the accidental murmurs were systolic in time, and 84 per cent of them were heard at the base of the heart.

3. Transmission of accidental murmurs is relatively rare. Nine and four-tenths per cent of the murmurs so classified were transmitted to the axilla, and only 0.8 per cent upward from the base. Cardiac hypertrophy was encountered in association with accidental murmurs in 8.6 per cent of the cases, but in every instance it was possible to account for the hypertrophy independently of the murmur. Cardiac hypertrophy is a necessary part of organic valvular disease.

4. Accentuation of the pulmonic second sound is heard in connection with accidental murmurs of the inorganic type, but is not to be expected with other types of these murmurs.

5. Other evidence than that afforded by a murmur must be found before a heart is assumed to be diseased.

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STATISTICAL STUDIES BEARING ON PROBLEMS IN THE CLASSIFICATION OF HEART DISEASES

V. HEART DISEASE AMONG EX-SERVICE MEN*†

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INTRODUCTION

THIS study was undertaken to ascertain the varieties of heart disease found among ex-service men; to classify these by etiology, structural lesion and functional capacity; and to attempt to analyze the cardiovascular findings in a group of men under hospitalization in the U. S. Veterans Bureau. While the average age of the World War veteran is approximately thirty-five, there are a considerable number of veterans of the Spanish American War, Philippine Insurrection, and other campaigns with an average age of fifty-four; the average age of all veterans is approximately thirty-six.

The data compiled and analyzed in this study were obtained by means of a questionnaire, which was issued to all of the U. S. Veterans hospitals and was executed on all of the Bureau beneficiaries under hospitalization for heart disease during 1926. The number of beneficiaries was 736.

These patients were under treatment in some 53 hospitals, and their cardiovascular diseases were studied and diagnoses made by Bureau physicians, some of whom have had no special training in heart disease. It would seem, therefore, that there might be a lack of uniformity in diagnostic criteria and in the diagnosis of heart disease. As the result of this it is possible that the classification of the heart lesions is not uniform, so that the data compiled herein may not be as valuable as if collected at a well-organized heart clinic.

It is thought, however, that these data on heart disease serve their purpose in that they constitute a composite study of this class of disease in a select group of individuals under hospitalization. The work having been done by a number of Bureau physicians has resulted in the absence of any inclinations toward certain personal diagnostic tendencies.

Much stress has been laid by a number of writers^{1, 2} upon the age of forty years as a period of the human life span when heart disease morbidity as well as mortality begins to mount. A study of heart

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disease at this time might contribute clinical as well as statistical information which would prove of value in any preventive measures in contemplation.

Dublin² states that five-sixths of the deaths from heart disease occur after the age of forty years, most of these being due to degenerative diseases and also arteriosclerosis. Cohn¹ contends that these deaths are not due to any definite disease processes, but, for the most part, to senescent changes of the heart and also to the fact that these subjects have been spared from earlier deaths resulting from infectious diseases and have succumbed to degenerative heart lesions.

As the result of this, Cohn, in a study of death rates by age groups, found a decided increase in the mortality rate from heart disease after the age of forty years. Under this age the death rate from heart disease has actually fallen.

INCIDENCE OF HEART LESIONS

Table I lists 1123 heart lesions found in 736 patients. The average number of lesions per patient is 1.53. It is noted that mitral insufficiency is the most common heart lesion recorded, followed by chronic fibrous myocarditis, hypertrophy of the heart, and aortic insufficiency, in the order named.

TABLE I

INCIDENCE OF HEART LESIONS IN A GROUP OF 736 EX-SERVICE MEN UNDER HOSPITALIZATION IN THE U. S. VETERANS BUREAU FOR HEART DISEASE

| HEART LESION | NUMBER | PER CENT |
|-------------------------------|--------|----------|
| Mitral insufficiency | 301 | 26.80 |
| Myocarditis, chronic, fibrous | 153 | 13.62 |
| Hypertrophy of heart | 148 | 13.18 |
| Aortic insufficiency | 111 | 9.88 |
| Mitral stenosis | 65 | 5.79 |
| Arteriosclerosis, general | 50 | 4.45 |
| Enlargement of heart | 41 | 3.65 |
| Aortitis without dilatation | 35 | 3.12 |
| Hypertension | 26 | 2.32 |
| Arrhythmia | 25 | 2.23 |
| Endocarditis, chronic | 21 | 1.87 |
| Aortitis with dilatation | 18 | 1.60 |
| Neurocirculatory asthenia | 17 | 1.51 |
| Auricular fibrillation | 17 | 1.51 |
| Tachycardia | 10 | 0.89 |
| Adherent pericardium | 9 | 0.80 |
| Arteriosclerosis, local | 8 | 0.71 |
| Myocarditis, acute | 7 | 0.62 |
| Aortic stenosis | 6 | 0.53 |
| Endocarditis, acute | 5 | 0.45 |
| Pericarditis with effusion | 4 | 0.36 |
| Atrophy of heart | 2 | 0.18 |
| Pulmonary stenosis | 2 | 0.18 |
| Hydropericardium | 2 | 0.18 |
| Heart-block | 2 | 0.18 |
| Tumor of pericardium | 1 | 0.09 |
| Pericarditis, fibrinous | 1 | 0.09 |
| Other lesions | 36 | 3.21 |
| Total | 1123 | 100.00 |

Reference to this table indicates that general arteriosclerosis occurred 50 times and constituted 4.45 per cent of the total lesions under observation. This is of interest in view of the fact that the average age of the Veterans Bureau beneficiaries at the time this study was made was thirty-six years.

ASSOCIATED CARDIOVASCULAR LESIONS

Table II is so arranged as to indicate the principal as well as one associated heart lesion. In this connection it is desirable to state that in a number of cases there was more than one associated heart lesion, but that it was possible to tabulate only one with the principal diagnosis.

In reviewing this table it is noted that 453 patients had but one cardiovascular lesion. The table also attempts to illustrate the incidence of some of the principal cardiac lesions, as well as their combinations.*

Dublin,² quoting Wyckoff and associates in the study of 1000 cases of organic heart disease, found that a number of the patients had two or more lesions. Of the total lesions observed, enlargement of the heart was found 884 times in association with other lesions and 203 times alone; mitral insufficiency 495 times; mitral stenosis 443 times; aortic insufficiency 146 times; aortic stenosis 29 times; aortitis 169 times; mitral insufficiency and mitral stenosis 421 times; mitral insufficiency, mitral stenosis, and aortic insufficiency 162 times; and mitral insufficiency, mitral stenosis, aortic insufficiency, and aortic stenosis 12 times.

Associated Cardiovascular Lesions With Mitral Insufficiency.—Table III is a list of 301 cases of mitral insufficiency so arranged as to indicate the associated cardiovascular lesions. It is noted that 167 of these cases had no coexisting lesion; 77 had but one coexisting lesion; the remaining 57 cases had 2 coexisting lesions associated with mitral insufficiency.† The largest group was a combination of mitral insufficiency, aortic insufficiency, and hypertrophy of the heart. It is therefore seen that mitral insufficiency and aortic insufficiency frequently coexist in the same patient.

*In determining the combinations of heart lesions in Table II it is first necessary to add the total number of lesions of each specific diagnosis found in the last column on the right to the total number of lesions of the same diagnosis at the bottom of the table; then subtract from this number the figure in the next to the last column, the latter indicating the number of times the particular heart lesion exists alone. The result of the above calculation is the number of times a particular lesion appears in combination. If it is desired to ascertain the number of times a lesion appears alone, the figures in the next to the last column only should be used.

†In determining the combinations of two coexisting heart lesions with the major diagnosis in Tables III, IV, V and VI, it is necessary to add the total number of lesions of each specific diagnosis found in the last column on the right to the total number of lesions of the same diagnosis at the bottom of the table; then subtract from this number the figure in the third column from the right, the latter indicating the number of times the particular heart lesion exists with but one coexisting lesion. If it is desired to ascertain the number of times a cardiovascular disease appears with one associated lesion, the figure in the third column from the right only should be used. It should be noted also that the figure in the second column from the right indicates the number of times a cardiovascular lesion appears alone.

TABLE II—CONT'D

| CARDIOVASCULAR DISEASE | CARDIOVASCULAR DISEASE |
|------------------------|---|
| 84 | CARDIAC VALVULAR DISEASE MITRAL INSUFFICIENCY |
| 26 | HYPERTROPHY OF HEART |
| 26 | CARDIAC VALVULAR DISEASE, MITRAL STENOSIS |
| 24 | MYOCARDITIS, CHRONIC |
| 23 | CARDIAC VALVULAR DISEASE, AORTIC INSUFFICIENCY |
| 20 | ARTERIOSCLEROSIS, GENERAL |
| 14 | AORTITIS, WITHOUT DILATATION |
| 12 | 1 |
| 10 | 1 |
| 9 | ARHYTHMIA |
| 6 | ENLARGEMENT OF HEART |
| 6 | ENDOCARDITIS, CHRONIC |
| 4 | AORTITIS, WITH DILATATION |
| 3 | PERICARDITIS, WITH EFFUSION |
| 3 | TACHYCARDIA |
| 2 | CARDIAC VALVULAR DISEASE, AORTIC STENOSIS |
| 2 | ADHERENT PERICARDIUM |
| 2 | ANEURYSM OF AORTA |
| 1 | MYOCARDITIS, ACUTE |
| 1 | HYDROPERICARDIUM |
| 1 | ANEURYSM OF AORTA, ASCENDING |
| 1 | ANEURYSM OF AORTIC ARCH |
| 1 | THROMBOPHLEBITIS, SAPHENOUS, RIGHT INTERNAL BRANCH |
| 1 | ARTERIOSCLEROSIS, CEREBRAL ARTERIES |
| 1 | HEART-BLOCK |
| 1 | ONLY ONE CARDIOVASCULAR LESION |
| 736 | TOTAL |

| | |
|--|--|
| Atrophy of heart | |
| Fatty degeneration of heart | |
| Congenital abnormality of the heart | |
| Thrombosis of left lenticulostriate artery | |
| Endocarditis, unclassified | |
| Cardiac valvular disease, pulmonic stenosis | |
| Pericarditis, fibrinous | |
| Pericarditis with effusion | |
| Tumor of pericardium | |
| Aneurysm of aorta | |
| Aneurysm of aorta, ascending | |
| Cardiac murmur, functional | |
| Thromboangiitis obliterans | |
| Dextrocardia | |
| Endarteritis obliterans | |
| Hypotension | |
| Aneurysm of left occipital artery | |
| Aneurysm of pulmonary artery | |
| Thrombosis of striate arteries, ext. and int. | |
| Thrombosis of right lenticulostriate artery | |
| Thrombosis of femoral, saphenous and popliteal veins | |
| Thrombosis of veins of pelvis and legs | |
| TOTAL | |

TABLE III
SHOWING 301 CASES OF MITRAL INSUFFICIENCY, TOGETHER WITH ASSOCIATED
CARDIOVASCULAR LESIONS

| CARDIOVASCULAR DISEASE | CARDIOVASCULAR DISEASE | | | | | | | | | | | | | TOTAL |
|---|--|---------------------------|----------------------|---------------------------|-----------------------|------------------------------|---|---------------------------|--------------|---------------|-------------|--|---|-------|
| | CARDIAC VALVULAR DISEASE, AORTIC INSUFFICIENCY | MYOCARDITIS, CHR. FIBROUS | HYPERTROPHY OF HEART | ARTERIOSCLEROSIS, GENERAL | ENDOCARDITIS, CHRONIC | AORTITIS, WITHOUT DILATATION | CARDIAC VALVULAR DISEASE, MITRAL STENOSIS | AORTITIS, WITH DILATATION | HYPERTENSION | AFIBRILLATION | HEART-BLOCK | ONLY ONE COEXISTING CARDIOVASCULAR DISEASE | MITRAL INSUFFICIENCY WITHOUT COEXISTING C. V. DISEASE | |
| Hypertrophy of heart | 12 | 5 | | 2 | 1 | | | | | | | | | 33 |
| Cardiac valvular disease, aortic insufficiency | | | | | | | 1 | 1 | | 1 | | | | 29 |
| Cardiac valvular disease, mitral stenosis | 4 | 2 | | | | | | | | | | | | 23 |
| Myocarditis, chr. fibrous | 1 | | 2 | 1 | 1 | | | | | | | | | 13 |
| Enlargement of heart | 1 | 1 | 2 | | | | | | | | | | | 8 |
| Endocarditis, chronic | 2 | | | | | | | | | | | | | 7 |
| Arteriosclerosis, general | | 1 | 3 | | | | | | 1 | | | | | 6 |
| Aortitis, without dilatation | 2 | 1 | | | | | | | | | | | | 3 |
| Aortitis, with dilatation | 1 | | | | | | | | | | | | | 3 |
| Aneurysm of aorta | 1 | | | | | | | | | | | | | 1 |
| Aneurysm of aorta, ascending | | | | | | | | | | | | | | 1 |
| Endocarditis, chronic | | | | | | | | | | | | | | 1 |
| Thrombophlebitis, right internal saphenous | 1 | | | | | | | | | | | | | 1 |
| Cardiac valvular disease, pulmonic stenosis | 1 | | | | | | | | | | | | | 1 |
| Hypertension | | | | | | | | | | | | | | 1 |
| Auricular fibrillation | | | | | | | | | | | | | | 1 |
| Tachycardia | | | | | | | | | | | | | | 1 |
| Arrhythmia | | | | | | | | | | | | | | 1 |
| Mitral insufficiency without coexisting c. v. disease | | | | | | | | | | | 2 | | | 2 |
| Total | 28 | 10 | 8 | 3 | 2 | 1 | 1 | 1 | 1 | 1 | 2 | 77 | 167 | 303 |

Associated Cardiovascular Lesions With Hypertrophy of Heart.—Table IV lists 148 cases of cardiac hypertrophy in such a manner as to show the various coexisting lesions. It is noted that 49 of these are cases of hypertrophy of heart without any other heart lesions; 39 had but one coexisting heart lesion, of this number 13 were cases of mitral insufficiency. Of the remaining 60 cases of hypertrophy of the heart having two coexisting heart lesions, the largest group is a combination of mitral insufficiency, aortic insufficiency, and hypertrophy of the heart.

Associated Cardiovascular Lesions With Chronic Myocarditis.—Table V lists 153 cases of chronic myocarditis arranged in such a manner as to show the various combinations of lesions. It is noted that 74 were cases of chronic myocarditis without any coexisting heart lesions; 43 cases of chronic myocarditis had but one coexisting lesion, of which number, that of chronic myocarditis with mitral insufficiency, was the largest group; 36 cases of chronic myocarditis had two coexisting heart lesions, the largest group of which was a combination of hypertrophy of the heart, enlargement of the heart, and chronic myocarditis.

Associated Cardiovascular Lesions With Aortic Insufficiency.—Table VI lists 111 cases of aortic insufficiency in such a manner as to show the various combinations of cardiovascular lesions. It is noted that of the total number, 19 are aortic insufficiency without any coexisting heart lesions; there are 52 cases of aortic insufficiency with but one coexisting lesion, the largest group of which is that of mitral insufficiency. Of those cases of aortic insufficiency with two coexisting heart lesions, 40 in number, the largest group is a combination of hypertrophy of the heart, mitral insufficiency, and aortic insufficiency.

ETIOLOGICAL TYPES OF HEART DISEASE

Wyckoff and Lingg³ in their study of the distribution of 1051 cases of heart disease found that 42.7 per cent were of the rheumatic type; 22.2 per cent of the arteriosclerotic type; 8.6 per cent were of the syphilitic type; 8.6 per cent belonged to other etiological types, and 17.8 per cent of the heart lesions were of unknown etiology.

Haven Emerson,⁴ after a review of the histories of 927 adults with heart disease, found the distribution of the causative factors as follows:

| | |
|-----------------------|------|
| Acute rheumatic fever | 331 |
| Acute tonsillitis | 207 |
| Carious teeth | 163 |
| Syphilis | 122 |
| Scarlet fever | 78 |
| Measles | 54 |
| Diphtheria | 47 |
| Pertussis | 14 |
| Chorea | 7 |
| Total | 1023 |

The above figures would indicate that more than one etiological factor was the cause of the heart lesions in a number of the patients.

TABLE IV
SHOWING 148 CASES OF HYPERTROPHY OF HEART, TOGETHER WITH ASSOCIATED
CARDIOVASCULAR LESIONS

| CARDIOVASCULAR DISEASE | CARDIOVASCULAR DISEASE | | | | | | | | | | | | | TOTAL |
|---|--|---------------------------|----------------------|---------------------------|--------------|---------------------------|--|---|------------|---------------------------|------------------------------------|--|---|-------|
| | CARDIAC VALVULAR DISEASE, MITRAL INSUFFICIENCY | ARTERIOSCLEROSIS, GENERAL | ENLARGEMENT OF HEART | AORTITIS, WITH DILATATION | HYPERTENSION | MYOCARDITIS, CHR. FIBROUS | CARDIAC VALVULAR DISEASE, AORTIC INSUFFICIENCY | CARDIAC VALVULAR DISEASE, MITRAL STENOSIS | ARRHYTHMIA | PATY INFLTRATION OF HEART | EMBOLISM OF MIDDLE CEREBRAL ARTERY | ONLY ONE COEXISTING CARDIOVASCULAR DISEASE | HYPERTROPHY OF HEART WITHOUT COEXISTING C. V. DISEASE | |
| Myocarditis, chr. fibrous | 5 | 1 | 7 | 1 | | 1 | 2 | | 1 | 1 | 1 | 6 | | 25 |
| Cardiac valvular disease, mitral insufficiency | 12 | 3 | 2 | 1 | | | | 2 | | | | 13 | | 21 |
| Cardiac valvular disease, aortic insufficiency | | 6 | | | 3 | 1 | | | | | | 5 | | 16 |
| Hypertension | 2 | | 1 | | | 1 | | | | | | 2 | | 12 |
| Endocarditis, chronic | 1 | | | | | | | | | | | 2 | | 8 |
| Enlargement of heart | | | | | | | | | | | | 2 | | 4 |
| Aortitis, without dilatation | | 2 | | | | | | | | | | 2 | | 2 |
| Cardiac valvular disease, mitral stenosis | | | | | | | | | | | | 1 | | 1 |
| Myocarditis, acute | | | | | | | | | | | | | | 1 |
| Arteriosclerosis, local | | | | | | | | | | | | | | 1 |
| Aneurysm of aorta | | | | 1 | | | | | | | | | | 1 |
| Aneurysm of aortic arch | | 1 | | | | | | | | | | | | 1 |
| Pericarditis, with effusion | | 1 | | | | | | | | | | | | 1 |
| Auricular fibrillation | | | | | | | | | 1 | | | | | 1 |
| Arrhythmia | | | | | | | | | | | | | | 1 |
| Hypertrophy of heart without coexisting c. v. disease | | | | | | | | | | | | 1 | | 1 |
| Total | 20 | 14 | 10 | 3 | 3 | 2 | 2 | 2 | 2 | 1 | 1 | 39 | 49 | 148 |

TABLE V
SHOWING 153 CASES OF CHRONIC MYOCARDITIS TOGETHER WITH ASSOCIATED CARDIOVASCULAR LESIONS

| CARDIOVASCULAR DISEASE | CARDIOVASCULAR DISEASE | | | | | | | | | | | | TOTAL |
|---|------------------------|--|----------------------|---------------------------------|--|--------------|------------|------------------------------|-------------------------|------------------------|--|--|-------|
| | HYPERTROPHY OF HEART | CARDIAC VALVULAR DIS- EASE, MITRAL INSUFFI- CIENCY | ENLARGEMENT OF HEART | AORTITIS, WITHOUT DILATATION | CARDIAC VALVULAR DIS- EASE, MITRAL STENOSIS | HYPERTENSION | ARRHYTHMIA | ARTERIOSCLEROSIS, GENERAL | ARTERIOSCLEROSIS, LOCAL | AURICULAR FIBRILLATION | ONLY ONE COEXISTING CARDIOVASCULAR DISEASE | CHRONIC MYOCARDITIS WITHOUT COEXISTING C. V. DISEASE | |
| Cardiac valvular disease, mitral insufficiency | 5 | | 1 | 1 | 2 | | | | | | 9 | | 19 |
| Enlargement of heart | 7 | | | | | 1 | | | | | 5 | | 12 |
| Hypertrophy of heart | | 1 | | | | 1 | | | | | 6 | | 8 |
| Arrhythmia | 1 | | | | | | | | | | 5 | | 7 |
| Auricular fibrillation | | | | | | | | | 1 | | 6 | | 6 |
| Arteriosclerosis, general | 1 | 1 | 1 | | | | | | | | 1 | | 5 |
| Cardiac valvular disease, aortic insufficiency | 2 | 1 | | 1 | | | | | | | 1 | | 5 |
| Aortitis, without dilatation | | | | | | | | | | | 1 | | 4 |
| Cardiac valvular disease, mitral stenosis | | | | | | | | | | 1 | | | 3 |
| Aortitis, with dilatation | | | | | | | | | | | 2 | | 2 |
| Fatty infiltration of heart | 1 | | | | | | | | | | 1 | | 1 |
| Endocarditis, chronic | 1 | 1 | | | | | | | | | | | 1 |
| Aneurysm of aortic arch | | | | | | | | | | | 1 | | 1 |
| Embolism of middle cerebral artery | 1 | | | | | | | | | | 1 | | 1 |
| Cardiac valvular disease, aortic stenosis | | | | | | | | | | | 1 | | 1 |
| Hypertension | | | | | | | | | | | 1 | | 1 |
| Heart-block | | | | | | | | | | | | | 1 |
| Tachycardia | | | | | | | | | | | 1 | | 1 |
| Chronic myocarditis without coexisting c.v. disease | | | | | | | | | | | | 74 | 74 |
| Total | 19 | 4 | 2 | 2 | 3 | 2 | 2 | 1 | 1 | 1 | 43 | 74 | 153 |

TABLE VI
SHOWING 111 CASES OF AORTIC INSUFFICIENCY TOGETHER WITH ASSOCIATED CARDIOVASCULAR LESIONS

| CARDIOVASCULAR DISEASE | CARDIOVASCULAR DISEASE | | | | | | | | | | | | | | | TOTAL | |
|--|------------------------|-----------------------|----------------------|----------|--|----------------------|---------------------------------|------------------------------|-------------------|------------------------------|---------------------|--|---|--------------|--|-------|---|
| | HYPERTROPHY OF HEART | ENDOCARDITIS, CHRONIC | MYOCARDITIS, CHRONIC | FIBROSIS | CARDIAC VALVULAR DIS- EASE, MITRAL STENOSIS | ENLARGEMENT OF HEART | AORTITIS, WITHOUT DILATATION | AORTITIS, WITH DILATATION | ANEURYSM OF AORTA | ANEURYSM OF CLINAR ARTERY | ENDOCARDITIS, ACUTE | CARDIAC VALVULAR DIS- EASE, MITRAL INSUFFI- CIENCY | CARDIAC VALVULAR DIS- EASE, PULMONIC STENOSIS | HYPERTENSION | ONLY ONE COEXISTING CARDIOVASCULAR DISEASE | | AORTIC INSUFFICIENCY WITHOUT COEXISTING C. V. DISEASE |
| Cardiac valvular disease, mitral insufficiency | 12 | 2 | 1 | 1 | 4 | 1 | 2 | 2 | 1 | 1 | 1 | | 1 | 1 | 28 | | 56 |
| Aortitis, without dilatation | | 1 | 1 | | | | | | | | | | | | 8 | | 10 |
| Cardiac valvular disease, mitral stenosis | | | 2 | | | | | | | 1 | | 1 | | | 5 | | 7 |
| Hypertrophy of heart | | | | | | | | | | | | | | | 3 | | 5 |
| Enlargement of heart | | 1 | | | | | | | | | | | | | 1 | | 3 |
| Cardiac valvular disease, aortic stenosis | | | | | | 1 | | | | | | | | | 2 | | 3 |
| Arteriosclerosis, general | | 1 | | | | | | | | | | | | | 1 | | 2 |
| Aortitis, with dilatation | 1 | | | | | | | | | | | | | | 1 | | 2 |
| Myocarditis, chronic | | | | | | | | | | | | | | | 1 | | 1 |
| Aneurysm of aorta | | 1 | | | | | | | | | | | | | 1 | | 1 |
| Arteriosclerosis, local | | | | | | | | | | | | | | | 1 | | 1 |
| Pericarditis, with effusion | | | | | | | | | | | | | | | 1 | | 1 |
| Aortic insufficiency without coexisting c. v. disease | | | | | | | | | | | | | | | 1 | | 1 |
| Total | 13 | 7 | 4 | 4 | 4 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 52 | 19 | 111 |

Acute rheumatic fever, acute tonsillitis, and carious teeth exceeded other causes of heart disease in the 927 cases referred to by Dr. Emerson.

In a statistical study of 3000 patients with heart disease in New England, some of whom were patients at the Massachusetts General Hospital and others were private patients seen in consultation, Paul D. White⁵ found that 54.5 per cent were of the rheumatic type. This observer maintains that the large percentage of rheumatic heart disease is due to climate, social and economic abnormalities, family susceptibility, and a probable mild contagious character of rheumatic fever. Coronary disease was the cause of heart lesions in 20.5 per cent of the cases; hypertension in 31 per cent; syphilis in 4 per cent; hyperthyroidism in 3 per cent; subacute bacterial endocarditis in 2 per cent; angina pectoris (hospital patients) in 9.5 per cent; angina pectoris (private patients) in 21 per cent; coronary thrombosis in from 3 to 6 per cent, and in 2.5 per cent of the cases the etiological factor was unknown.

In an analysis of 360 cases of valvular heart disease discharged from the U. S. Navy, Bloedorn and Roberts⁶ found the principal etiological factors as follows: Rheumatic fever was the etiological factor in 101 cases; tonsillitis in 59; syphilis in 3; pneumonia in 4, and influenza in 4.

Wycoff and Lingg,³ in a series of 499 cases of heart disease, found that 59.7 per cent were due to acute rheumatic fever and 12.7 per cent were due to tonsillitis. It is noted that in the group of cases studied by these observers no case of heart disease was attributed to diseases of the teeth and gums.

Table VII is a classification of heart disease found among 736 Bureau beneficiaries into 11 types of etiological factors, similar to that used by the American Heart Association. Attention is invited to the fact that in a number of cases two or more etiological factors

TABLE VII

ETIOLOGICAL TYPES OF HEART DISEASE IN 736 BUREAU PATIENTS WITH 1761
ETIOLOGICAL FACTORS, SHOWING PER CENT OF TOTAL NUMBER
WITHIN EACH TYPE

| ETIOLOGICAL TYPE | NUMBER | PER CENT |
|---------------------------|--------|----------|
| Rheumatic | 590 | 33.50 |
| Other infectious diseases | 529 | 30.04 |
| Syphilitic | 197 | 11.19 |
| General systemic disease | 76 | 4.32 |
| Arteriosclerotic | 71 | 4.03 |
| Toxic | 35 | 1.99 |
| Traumatic | 34 | 1.93 |
| Thyroid | 23 | 1.31 |
| Neurogenic | 15 | 0.85 |
| Others | 45 | 2.55 |
| Unknown | 146 | 8.29 |
| Total | 1761 | 100.00 |

were present, making a total of 1761 factors in a series of 1123 lesions. The average number of etiological factors per lesion was 1.56, and the average number of etiological factors per patient was 2.39.

A review of Table VII indicates that the principal etiological factors of heart disease as found in a series of ex-service men of an average age of thirty-six are: rheumatism, other infectious diseases, and syphilis. Attention is invited to the fact that arteriosclerosis was an etiological factor to the extent of 4.03 per cent only.

Rheumatic Heart Disease.—Table VIII lists rheumatic heart disease according to the types of infection. It is noted that tonsillitis, acute rheumatic fever, and tooth and gum infections constitute the principal etiological factors in 83.22 per cent of a series of 590 cases of rheumatic heart disease.

The incidence of the other etiological rheumatic factors was of minor importance. Reference to Table VIII shows that they comprise only 99, or 16.78 per cent, of the total number of rheumatic factors in the series of cases studied.

TABLE VIII

RHEUMATIC HEART DISEASE BY TYPE OF INFECTION SHOWING PER CENT OF EACH TYPE

| TYPE OF INFECTION | NUMBER | PER CENT |
|--------------------------|--------|----------|
| Tonsillitis | 238 | 40.34 |
| Acute rheumatic fever | 131 | 22.20 |
| Tooth and gum infections | 122 | 20.68 |
| Joint pains | 35 | 5.93 |
| Muscle pains | 19 | 3.22 |
| Pharyngitis | 11 | 1.86 |
| Arthritis | 11 | 1.86 |
| Rheumatic nodules | 8 | 1.36 |
| Chorea | 8 | 1.36 |
| Osteoarthritis | 4 | 0.68 |
| Torticollis | 1 | 0.17 |
| Diseased adenoids | 1 | 0.17 |
| Myositis | 1 | 0.17 |
| Total | 590 | 100.00 |

Heart Disease, the Sequel of Infectious Diseases.—While the part played by the infectious diseases as etiological factors of heart disease is based upon the histories of the cases as obtained from the patients, in a large number the causative infectious disease was incurred in the military service, and was followed by the heart lesion reported on the questionnaire. Most of the data are on record and therefore dependable.

A study of the data recorded in this paper indicates that the other infectious diseases constitute a group of etiological factors of heart disease second only to the rheumatic infections.

Whether heart lesions due to infectious diseases are latent for a long period and become clinically apparent only some time after the

occurrence of the infectious disease is yet to be ascertained. According to data in Table IX, one would infer that such is a possibility.

Table IX lists the infectious diseases which were considered etiological factors of heart disease in this study. It is noted that the incidence of influenza as an etiological factor of infectious heart disease was very great—in fact it was given as the cause of heart disease in 167, or 31.57 per cent, of a series of 529 cases.

Some of the other principal causative infectious diseases were: pneumonia, tuberculosis, measles, typhoid fever, gonorrhea and otitis media.

TABLE IX

HEART DISEASE, THE SEQUEL OF INFECTIOUS DISEASES, BY TYPE OF DISEASE

| INFECTIOUS DISEASE | NUMBER | PER CENT |
|--------------------------|--------|----------|
| Influenza | 167 | 31.57 |
| Pneumonia | 85 | 16.07 |
| Tuberculosis | 75 | 14.18 |
| Measles | 42 | 7.94 |
| Typhoid fever | 32 | 6.05 |
| Gonorrhea | 31 | 5.86 |
| Otitis media | 28 | 5.29 |
| Scarlet fever | 25 | 4.73 |
| Diphtheria | 18 | 3.40 |
| Dysentery | 16 | 3.02 |
| Mumps | 5 | 0.94 |
| Amebiasis | 2 | 0.38 |
| Cerebrospinal meningitis | 1 | 0.19 |
| Malaria | 1 | 0.19 |
| Dengue | 1 | 0.19 |
| Total | 529 | 100.00 |

INTERVAL BETWEEN OCCURRENCE OF ETIOLOGICAL FACTOR AND APPEARANCE OF CARDIOVASCULAR DISEASE

Haven Emerson⁴ in an analysis of 100 cases of rheumatic heart disease for the purpose of ascertaining the length of time elapsing between the date of probable infection and knowledge of cardiac disease found that the average period was 5.36 years. The periods varied widely from patients where knowledge was immediate at the time of the acute sickness to those who had evidently had their disease thirty years before knowledge of it was obtained.

Emerson also found that in the case of 23 patients with syphilitic heart disease the average time before knowledge of the cardiac disease was obtained was 17.65 years, the range being from one to thirty-four years.

Cohn, quoting Mackie,⁷ says that among 25 cases of mitral stenosis the lesion was developed within twenty-four months in 22 cases, but that it required five and a half, six and a half and five and two-thirds years in the 3 additional cases. It may then take from one to six years for the cardiac lesions to develop; meanwhile manifestations of rheumatic disease may be quite absent.

Table X illustrates the interval between the operation of the etiological factors and the recognition of the cardiovascular lesions in a series of 736 patients. In considering the entire group of 1761 etiological factors, it is noted that in 886 instances it was impossible to determine the interval between the occurrence of the etiological factor and the appearance of the cardiovascular disease. Of the remainder, 875 in number, the physicians were able to record the time interval. Of this number, in 108 instances the heart lesion developed immediately; in 26 instances within one month; in 59 instances between one and three months; in 34 between three and six months; in 21 between six and twelve months; in 325 between one and five years; in 229 between five and ten years; and in 73 instances between ten and twenty-five years.

It is believed that these data when critically analyzed are subject to question. What the mechanism is in the production of a heart lesion a number of years after the occurrence of the infection is not known. Perhaps the heart valve is affected at the time of the appearance of the disease without giving any clinical evidence. These figures, therefore, should be interpreted as indicating the time between the occurrence of the etiological disease and the clinical recognition of the heart lesion.

Further analysis of the data in Table X would lead one to assume that the interval of time elapsing between the occurrence of the etiological disease and the appearance of the cardiac lesion is dependent upon several factors: first, the type of causative disease; second, extent of degenerative changes and location of heart lesion; and finally, the rapid or slow development of cardiac lesions is dependent upon the amount of functional activity to which the heart is subjected. If the heart lesion is slight, it is thought the damage might not be sufficient to bring about abnormal cardiac function; if the latter becomes apparent, it would be some time after the occurrence of the causative disease. On the other hand, if the heart which had undergone certain abnormal changes, even though slight, were subjected to excessive physical strain, cardiac derangement sufficient to be recognized clinically might appear early.

DURATION OF CARDIOVASCULAR DISEASE

Any discussion of the duration of heart disease in ex-service men must be cautious, because of the fact that a great many of the patients attribute the heart disease to increased physical effort or to diseases incurred while in the military service. This is to be expected. While in the majority of instances the data are reliable, in some, the findings may be questionable. However, in spite of this, the figures in Table XI give an approximate idea of the duration of cardiovascular disease

TABLE X
INTERVAL BETWEEN OCCURRENCE OF ETIOLOGICAL FACTOR AND APPEARANCE OF CARDIOVASCULAR DISEASE IN 736 PATIENTS WITH 1761
ETIOLOGICAL FACTORS

| ETIOLOGICAL FACTOR | IMMEDI- ATELY | LESS THAN 1 MO. | 1 TO 3 MO. | 3 TO 6 MO. | 6 TO 12 MO. | 1 TO 2 YR. | 2 TO 3 YR. | 3 TO 4 YR. | 4 TO 5 YR. | 5 TO 6 YR. | 6 TO 7 YR. | 7 TO 8 YR. | 8 TO 10 YR. | 10 TO 25 YR. | UN- KNOWN INTERVAL | TOTAL | PER CENT |
|-------------------------------|------------------|-----------------------|---------------|---------------|----------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|----------------|--------------------|--------------------------|-------|-------------|
| Rheumatic | 49 | 15 | 23 | 15 | 14 | 53 | 21 | 19 | 18 | 10 | 18 | 5 | 36 | 31 | 263 | 590 | 33.50 |
| Infectious diseases | 29 | 10 | 27 | 16 | 7 | 64 | 20 | 20 | 17 | 12 | 14 | 7 | 26 | 31 | 229 | 529 | 30.04 |
| Syphilitic | 3 | | 4 | 2 | | 10 | 5 | 4 | | 4 | 9 | 7 | 42 | 2 | 105 | 197 | 11.19 |
| General systemic dis- ease | 10 | | | 1 | | 12 | 10 | 2 | 3 | | 4 | | 2 | | 32 | 76 | 4.32 |
| Arteriosclerotic | 3 | | | | | 6 | 6 | | 3 | | 5 | | 3 | 1 | 44 | 71 | 4.03 |
| Toxic | 3 | 1 | | | | 1 | | 1 | 3 | 3 | 2 | 1 | 6 | | 17 | 35 | 1.99 |
| Traumatic | 6 | | 1 | | | 2 | 3 | 2 | 2 | | 2 | | 2 | 1 | 13 | 34 | 1.93 |
| Thyroid | | | 3 | | | 4 | 2 | | | | | 1 | 3 | | 10 | 23 | 1.31 |
| Neurogenic | 3 | | 1 | | | 3 | | | | 2 | 1 | | | 1 | 6 | 15 | 0.85 |
| Others | 2 | | | | | 3 | 6 | 3 | | | | | 2 | 6 | 21 | 45 | 2.55 |
| Unknown factor | | | | | | | | | | | | | | | 146 | 146 | 8.29 |
| Total | 108 | 26 | 59 | 34 | 21 | 158 | 73 | 51 | 43 | 31 | 55 | 21 | 122 | 73 | 886 | 1761 | 100.00 |

in these 736 patients. It is noted that of the total number it was impossible to determine the duration of heart disease in 245 cases. In the remainder, namely, 491 cases, heart disease existed for periods varying from one to six months in 30 cases to from eight to nine years in 41 cases; in 3 cases the heart disease was congenital; the largest group, 85 in number, gave a history of having had heart disease from seven to eight years.

TABLE XI

TABLE SHOWING DURATION OF CARDIOVASCULAR DISEASE IN A GROUP OF 736 PATIENTS

| | NUMBER OF CASES |
|----------------|-----------------|
| Congenital | 3 |
| 1 to 6 months | 30 |
| 6 to 12 months | 47 |
| 1 to 2 years | 31 |
| 2 to 3 years | 51 |
| 3 to 4 years | 29 |
| 4 to 5 years | 47 |
| 5 to 6 years | 54 |
| 6 to 7 years | 73 |
| 7 to 8 years | 85 |
| 8 to 9 years | 41 |
| Time not known | 245 |
| Total | 736 |

Table XII gives a list of the specific heart diseases in a series of 255 of the total number of 736 patients. These patients were hospitalized primarily for heart disease.

In the consideration of these 255 cases it is realized, of course, that the date of diagnosis of the heart disease was not necessarily the date of its inception. For instance, the duration of chronic myocarditis, combined aortic and mitral disease, and of mitral insufficiency, as indicated in Table XII, is very much shorter than is the customary clinical experience. It is thought that in a number of these cases the heart lesions were present some time before they were recognized clinically. The figures, however, serve their purpose in showing the clinical duration of certain cardiovascular lesions among ex-service men hospitalized by the United States Veterans Bureau.

A study was also made of the duration of various cardiovascular lesions in 252 Bureau beneficiaries who died from heart disease. The time from the date of diagnosis of the lesion to the date of death was as follows:

For acute dilatation of the heart—eight months; acute endocarditis—four months; chronic endocarditis—four years; cerebral hemorrhage—two years; acute myocarditis—two years; chronic myocarditis—four years; combined aortic and mitral disease—five years; and mitral insufficiency—three years.

TABLE XII
DURATION OF CARDIOVASCULAR DISEASE IN 255 OF 736 PATIENTS SHOWING SPECIFIC CARDIOVASCULAR DIAGNOSIS

| CARDIOVASCULAR DISEASE | 1 TO 6 MO. | 6 MO. TO 1 YR. | 1 TO 2 YR. | 2 TO 3 YR. | 3 TO 4 YR. | 4 TO 5 YR. | 5 TO 6 YR. | 6 TO 7 YR. | 7 TO 8 YR. | 8 TO 9 YR. | UN- KNOWN | NOT RE- PORTED | TOTAL |
|--|---------------|-------------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|--------------|-------------------|-------|
| Myocarditis, chronic | 3 | 6 | 5 | 6 | 1 | 5 | 6 | 7 | 9 | 4 | 6 | 1 | 59 |
| Valvular heart disease, combined lesions, aortic and mitral | | 2 | 3 | 2 | 2 | 7 | 7 | 8 | 10 | 5 | 3 | | 49 |
| Valvular heart disease, mitral in- sufficiency | 1 | 2 | | 3 | 1 | 3 | 7 | 9 | 9 | 4 | 6 | | 45 |
| Valvular heart disease, aortic in- sufficiency | 1 | 1 | 1 | 2 | 3 | 3 | 2 | 2 | 4 | 1 | 1 | | 21 |
| Valvular heart disease, mitral stenosis | | 3 | 1 | 3 | 2 | 1 | 2 | 7 | 1 | 1 | 2 | 1 | 20 |
| Aortitis | | | | | 3 | | | | 1 | 1 | 2 | 1 | 7 |
| Asthenia, neurocirculatory | | | | | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 1 | 7 |
| Auricular fibrillation | | | | | 1 | 1 | 1 | 1 | 2 | | 1 | | 7 |
| Aneurysm, aorta | | 1 | | 1 | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 6 |
| Hypertension | | | | 1 | 1 | | 1 | | 1 | | 2 | | 6 |
| Arteriosclerosis, general | | | | | 1 | | | | 2 | | 1 | | 4 |
| Cardiac arrhythmia, otherwise un- classified | | | 1 | 1 | | | 1 | 2 | 1 | | 1 | | 3 |
| Cardiac hypertrophy | | | | | | | 1 | | | | | | 3 |
| Endocarditis, septic | | 1 | | | | | | | | | 1 | 1 | 3 |
| Arteriosclerosis, cerebral | | | 1 | 1 | | | | | 1 | 1 | | | 2 |
| Cardiac hypertrophy and dilatation | | | | | | | | | 1 | | 1 | | 2 |
| Myocarditis, acute | 1 | | | | | | | | | | | | 2 |
| Pericarditis, with effusion | 1 | 1 | | 1 | | | 1 | | 1 | | | | 2 |
| Tachycardia | | | | | | | | | | | | | 2 |
| Dilatation, aortic arch | | | | | | | | | | | | | 2 |
| Endocarditis, chronic | | | | | | | 1 | | 1 | | | | 1 |
| Heart-block | | | | | | | 1 | | | | | | 1 |
| Valvular heart disease, aortic stenosis | | | | | | | | | | | 1 | | 1 |
| Valvular heart disease, otherwise unclassified | | | | | | | | | | | | | 1 |
| Total | 7 | 17 | 10 | 20 | 14 | 21 | 30 | 38 | 45 | 18 | 30 | 5 | 255 |

These 252 beneficiaries were not part of the group of 736 cases previously referred to, but were a group arbitrarily selected for the purpose of ascertaining the period of time elapsing between the date of diagnosis of heart disease and date of death. Attention is invited to the fact that the duration of the various heart lesions is shorter than is the usual experience. The figures are, however, the actual clinical experience in the case of these 252 ex-service patients.

FUNCTIONAL CAPACITY IN CARDIOVASCULAR DISEASE

The statistics appearing in Table XIII consist of the data received from the various Bureau hospitals. The classification of the functional capacity of ex-service patients with cardiovascular disease is in accordance with the standards recommended by the American Heart Association.

In the consideration of the total number of patients it is noted that 209, or 28.40 per cent, were able to carry on their habitual physical activity; 218, or 29.62 per cent, were able to carry on a slightly diminished activity, and were therefore placed in Class 2-A; 142, or 19.29 per cent, were able to carry on a greatly diminished activity, and were therefore grouped in Class 2-B; whereas 82, or 11.14 per cent, were unable to carry on any physical activity, were confined to bed, and were grouped in Class 3; in 85, or 11.55 per cent, of the patients the functional capacity was not stated.

It is seen, therefore, that 442, or 60.05 per cent, of the whole group of 736 patients had various heart lesions which resulted in a reduced functional activity. These patients were classified as belonging to Groups 2-A, 2-B, and 3—such classification denotes that their earning capacity is impaired.

The Bureau has, therefore, a twofold problem to solve: first, how best to rehabilitate these patients physically; second, how to restore them to a state of maximum economic efficiency. The physical rehabilitation can be accomplished by the excellent hospital facilities of the Bureau available throughout the country, where the patients with decompensation may be treated and restored to efficient cardiac function. This can be supplemented by the utilization of the dispensary facilities in the regional offices where these patients may be further treated and observed until they attain maximum cardiac improvement.

During hospitalization these cardiac patients may be instructed in various light occupational crafts, and an attempt could be made to ascertain the maximum physical effort which may be expended by the patient as well as his work tolerance. The occupations prescribed by the physician as therapy in the hospital can be so arranged as to assist in determining the vocational potentiality of the patient.

Upon restoration of the patient to normal cardiac function, the physician as well as the occupational therapy aide and also the social worker in the hospital should study the individual case for the purpose of ascertaining suitable vocational placement. It is important that the Bureau supervise closely the employment of these cardiac cases after their discharge from hospitalization. These patients should be urged to return periodically either to the out-patient department of the hospital or to the dispensary of the regional office for cardiac examinations, so that the effect of work upon the circulatory system may be determined. If it is found that the physical effort expended in this direction is causing decompensation, the physician as well as the social worker should urge the patient to change the character, or the amount, of work.

It is believed that by such a procedure not only would the lives of many of these patients be prolonged and saved, but, in many instances, hospitalization would be resorted to less frequently and the amount of financial compensation might be reduced because of a decrease of the occupational handicap and an increase in work tolerance.

The above views are predicated upon that section of the World War Veterans Act which states that "The Bureau will have the power and it will be its duty to provide for the placement of rehabilitated persons in suitable and gainful occupations." Based on the above, the Director of the U. S. Veterans Bureau wisely promulgated a letter dated December 14, 1927, to regional managers. He stated therein that it was his desire that contacts be established with a view of assisting veterans in obtaining employment. He requested that a close cooperation be established with civilian agencies for the purpose of placing disabled ex-service men in suitable and gainful occupations.

The Director in issuing this letter evidently felt that the responsibility of the Bureau did not cease with the hospitalization and compensation of these disabled ex-service men, but that the facilities of the Bureau should be extended still further with a view of prolonging the lives of these patients and guiding them through occupational hazards. This, if properly done, will result not only in direct benefit of patients, but indirectly in an economic saving to the government.

EFFECT OF TONSILLECTOMY ON HEART DISEASE

In view of the fact that tonsillitis was considered an etiological factor in 238, or 40.3 per cent, of 590 cases of rheumatic heart disease, it was thought that a study should be made of the effect of tonsillectomy upon the course of the disease and upon the final outcome of the case.

TABLE XIII
SHOWING FUNCTIONAL CAPACITY OF 736 EX-SERVICE PATIENTS WITH CARDIOVASCULAR DISEASE, ACCORDING TO THE CLASSIFICATION OF THE AMERICAN HEART ASSOCIATION

| | ABLE TO CARRY ON HABITUAL PHYSICAL ACTIVITY (1) | ABLE TO CARRY ON SLIGHTLY DIMINISHED PHYSICAL ACTIVITY (2A) | ABLE TO CARRY ON GREATLY DIMINISHED PHYSICAL ACTIVITY (2B) | UNABLE TO CARRY ON ANY PHYSICAL ACTIVITY (3) | NOT STATED | TOTAL | PER CENT |
|-------------------------------------|---|--|---|--|---------------|-------|----------|
| Mitral insufficiency | 84 | 72 | 24 | 8 | 7 | 195 | 26.49 |
| Myocarditis, chronic, fibrous | 21 | 36 | 28 | 18 | 16 | 119 | 16.16 |
| Hypertrophy of heart | 26 | 37 | 24 | 13 | 9 | 109 | 14.81 |
| Aortic insufficiency | 19 | 25 | 18 | 11 | 10 | 83 | 11.27 |
| Enlargement of heart | 5 | 5 | 11 | 5 | 8 | 34 | 4.62 |
| Mitral stenosis | 6 | 7 | 5 | 6 | 9 | 33 | 4.48 |
| Arteriosclerosis, general | 7 | 5 | 5 | 2 | 1 | 20 | 2.71 |
| Neurocirculatory asthenia | 4 | 3 | 3 | 2 | 7 | 17 | 2.31 |
| Endocarditis, chronic | 4 | 3 | 4 | 2 | 1 | 14 | 1.90 |
| Aortitis, without dilatation | 6 | 4 | 4 | 2 | 2 | 14 | 1.90 |
| Arrhythmia | 6 | 3 | 3 | 2 | | 12 | 1.63 |
| Aortitis, with dilatation | 3 | 3 | 1 | 2 | | 9 | 1.22 |
| Adherent pericardium | 3 | 2 | 2 | | | 7 | 0.95 |
| Hypertension | 4 | 2 | 1 | | | 7 | 0.95 |
| Tachycardia | 3 | 2 | 1 | | | 7 | 0.95 |
| Myocarditis, acute | | 1 | 2 | 2 | 1 | 6 | 0.81 |
| Arteriosclerosis, cerebral arteries | 2 | 1 | 1 | 1 | | 5 | 0.68 |
| Endocarditis, acute | | | | 1 | 4 | 5 | 0.68 |
| Auricular fibrillation | 1 | 2 | | 1 | | 4 | 0.54 |
| Pericarditis, unclassified | 1 | 1 | 2 | | | 4 | 0.54 |
| Aortic stenosis | | 2 | | 1 | | 3 | 0.40 |

TABLE XIII—Cont'd

| | ABLE TO CARRY ON HABITUAL PHYSICAL ACTIVITY (1) | ABLE TO CARRY ON SLIGHTLY DIMINISHED PHYSICAL ACTIVITY (2A) | ABLE TO CARRY ON GREATLY DIMINISHED PHYSICAL ACTIVITY (2B) | UNABLE TO CARRY ON ANY PHYSICAL ACTIVITY (3) | NOT STATED | TOTAL | PER CENT |
|---|---|--|---|--|---------------|-------|-------------|
| Aneurysm of aortic arch | 2 | | | 3 | | 3 | 0.40 |
| Atrophy of heart | | 1 | | | 1 | 2 | 0.27 |
| Congenital abnormality of heart | | | | | 2 | 2 | 0.27 |
| Fatty degeneration of heart | | | 1 | 1 | | 2 | 0.27 |
| Thrombosis of left lenticulostriate artery | | | 1 | | | 1 | 0.14 |
| Thrombosis of right lenticulostriate artery | | | | | 1 | 1 | 0.14 |
| Cardiac murmur, not organic | | | 1 | | | 1 | 0.14 |
| Dextrocardia | | | | 1 | | 1 | 0.14 |
| Pericarditis, fibrinous | | | | 1 | | 1 | 0.14 |
| Pericarditis, with effusion | | | | 1 | | 1 | 0.14 |
| Pulmonic stenosis | 1 | | | | | 1 | 0.14 |
| Tumor of pericardium | | | | | 1 | 1 | 0.14 |
| Aneurysm of aorta | | | | | 1 | 1 | 0.14 |
| Aneurysm of aorta, ascending | | | 1 | | | 1 | 0.14 |
| Aneurysm of left occipital artery | 1 | | | | | 1 | 0.14 |
| Aneurysm of pulmonary artery | | | | | 1 | 1 | 0.14 |
| Thrombosis of striate arteries, external and internal | | | | 1 | | 1 | 0.14 |
| Thrombosis of femoral, saphenous, and popliteal veins | | | | | 1 | 1 | 0.14 |
| Thrombosis of veins of pelvis and leg | | | | | 1 | 1 | 0.14 |
| Endarteritis obliterans | | | 1 | | | 1 | 0.14 |
| Endocarditis, unclassified | | | 1 | | | 1 | 0.14 |
| Hypotension | | 1 | | | | 1 | 0.14 |
| Thromboangiitis obliterans | | | 1 | | | 1 | 0.14 |
| Total | 209 | 218 | 142 | 82 | 85 | 736 | 100.00 |

A number of observers have made similar studies of this problem and the conclusions reached have been at variance with each other.

Cohn² refers to 391 patients with rheumatic manifestations on whom tonsillectomy was done, with subsequent observations. The rheumatic manifestations for which the operation was performed recurred in 49.6 per cent of the cases.

In 175 patients with rheumatic manifestations on whom tonsillectomy was not done, 111, or 64 per cent, suffered from a recurrence of the disease. Cohn finds from a study of the cases reported by certain observers that, on the whole, it cannot be said that the effect upon rheumatic fever of the removal of the tonsils is satisfactory, inasmuch as recurrence of rheumatic manifestations takes place in 50 per cent of the cases.

St. Lawrence⁸ observed 58 cases of organic cardiac disease before and after tonsillectomy and found that, in general, there seemed to be a fairly prompt increase both in desire and in capacity for exertion; and the exercise tolerance seemed to be favorably influenced more often, more promptly, and to a greater degree by tonsillectomy than by any other measure utilized in the care of cardiac children. In a series of 5 cases it was observed that an incomplete removal of the tonsils had little or no effect on the recurrence, while complete tonsillectomy was followed by a cessation of the symptoms of heart disease.

Crowe, Watkins, and Rothholz, quoted by Cohn,⁹ noted recurrences of rheumatic fever in 4 out of 25 cases under observation; these occurred from three weeks to three years after tonsillectomy. In all of the 25 cases, except 4, there were cardiac lesions present; mitral lesions were present in 21; some of the patients had 2 or more coexisting cardiac lesions.

Of 24 cases of chorea with concomitant heart lesions, in 12 cases the same authors report that chorea recurred or failed to disappear in 11 instances ranging from eight months to three and one-sixth years after tonsillectomy.

A study of the Bureau statistics revealed the fact that tonsillectomy was done on 220 out of the total number of 736 patients under observation. One hundred and thirty-seven of this number had heart lesions prior to tonsillectomy; in 44 instances the heart lesions were first noted after the removal of the tonsils. In 15 the time of the first appearance of the heart lesion was unknown; and in 24 instances no report was made.

A review of the available data further shows that in 145 cases it was the opinion of the Bureau physicians that the cardiovascular disease was due primarily to tonsillitis, and that tonsillectomy had the following effect:

| | |
|--------------------|----|
| Improved | 18 |
| Retrogressed | 7 |
| No change | 28 |
| Undetermined | 7 |
| Not stated | 85 |

The above figures would indicate that the Bureau physicians found it difficult, in a number of instances, to attribute to the tonsillectomy any effect upon the cardiovascular disease. However, in 18, or 12.4 per cent, of the cases it was thought that improvement followed the operation. This estimate it is believed is conservative. It would appear, therefore, that if infected tonsils are an etiological factor of heart disease, their removal is followed by an improvement in a small percentage of cases.

(To be continued in April.)

ELECTROCARDIOGRAPHIC STUDIES ON THE ACTIONS OF DRUGS

I. THE VAGUS IN ETHER ANESTHESIA*

HARRY GOLD, M.D.

WITH

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IN COLLABORATION

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THE present study was undertaken to determine the influence of ether in the various stages of anesthesia upon the response of the vagus (cardio-inhibitory nerve) to stimulation by morphine.

Fifteen experiments were carried out on dogs, only those animals that were found to remain quiet without anesthesia or restraint being employed. Ether was given by inhalation (open cone), always with sufficient air to avoid asphyxial changes. Electrocardiograms (Lead II only was used) were taken at frequent intervals, and changes in the sinus rate and conduction time (P-R intervals) were noted. Since there are frequently considerable differences in the length of the P-R interval from beat to beat, ten such intervals in a given strip were determined, and the average was taken as the interval for that observation. Where several experiments were performed on the same dog a number of days intervened between them. Observations made in five of the experiments will be given in detail, as they suffice to illustrate the general results.

In the first experiment a dog (A) received 1 mg. of morphine sulphate per kg. intravenously, and within twenty-seven minutes the sinus rate had decreased from 180 to 80 a minute and the P-R interval had increased from 0.093 to 0.108 second, and there were some dropped ventricular groups. During the early stage of ether anesthesia nearly all the morphine effects disappeared and remained in abeyance during the period of deep ether narcosis. The T-wave showed interesting changes. In the control period it was negative and 1 mm. deep. During the morphine action it became 3 mm. deep. During the early stage of ether inhalation the morphine effect upon the T-wave persisted. In the stage of deep ether narcosis (corneal reflexes abolished, pupils dilated and not reacting to light, complete muscular relaxation, respiration good), the morphine effect upon the T-wave disappeared and the latter assumed the form present in the control period. Ether,

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therefore, in this experiment completely abolished the effects of morphine stimulation of the vagus. (See Figs. 1 and 2.)

In the second experiment the order was reversed, the same animal being anesthetized to deep ether narcosis before the morphine was administered. Ether increased the heart rate markedly and shortened the P-R interval. These effects established during the excitement stage of ether action persisted during the period of deep narcosis. Morphine sulphate (1 mg. per kg. intravenously) abolished the ether effect upon the heart rate and conduction, both factors returning to the level of

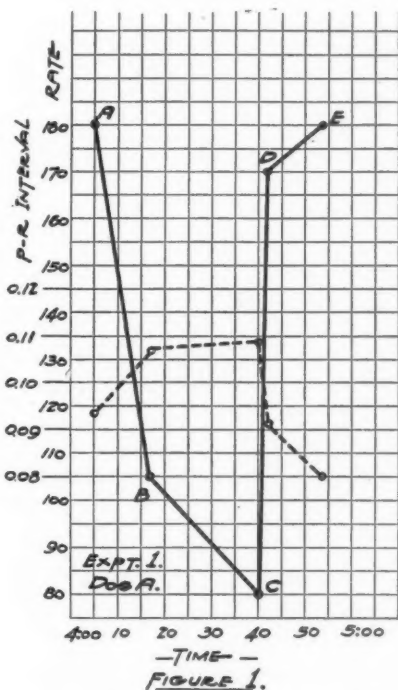


Fig. 1.—A, Control; B, five minutes after 1 mg. morphine sulphate per kg. intravenously, dropped ventricular groups; C, dropped ventricular groups; D, two minutes after starting inhalation of ether, struggling; E, deep ether anesthesia.

the control tracing, and during one-half hour of deep ether narcosis morphine produced no further effect. This is in striking contrast to the intensity of the effect of the same dose of morphine in the same dog in the first experiment, without ether. A second dose of morphine (2 mg. per kg. intravenously) caused a further decrease in the heart rate and increase in P-R interval, but the changes were not nearly so marked as those produced by one-half the dose of morphine in the same dog without ether. With the recovery from the ether the morphine effects became more pronounced. This experiment, therefore, shows that in the presence of ether narcosis morphine may produce

some vagal effects but much less intense than in the unanesthetized animal. (See Fig. 3.)

The third experiment shows essentially the same phenomena as the first. A second dose of morphine was given which produced primary acceleration of the heart rate and shortening of the P-R interval.

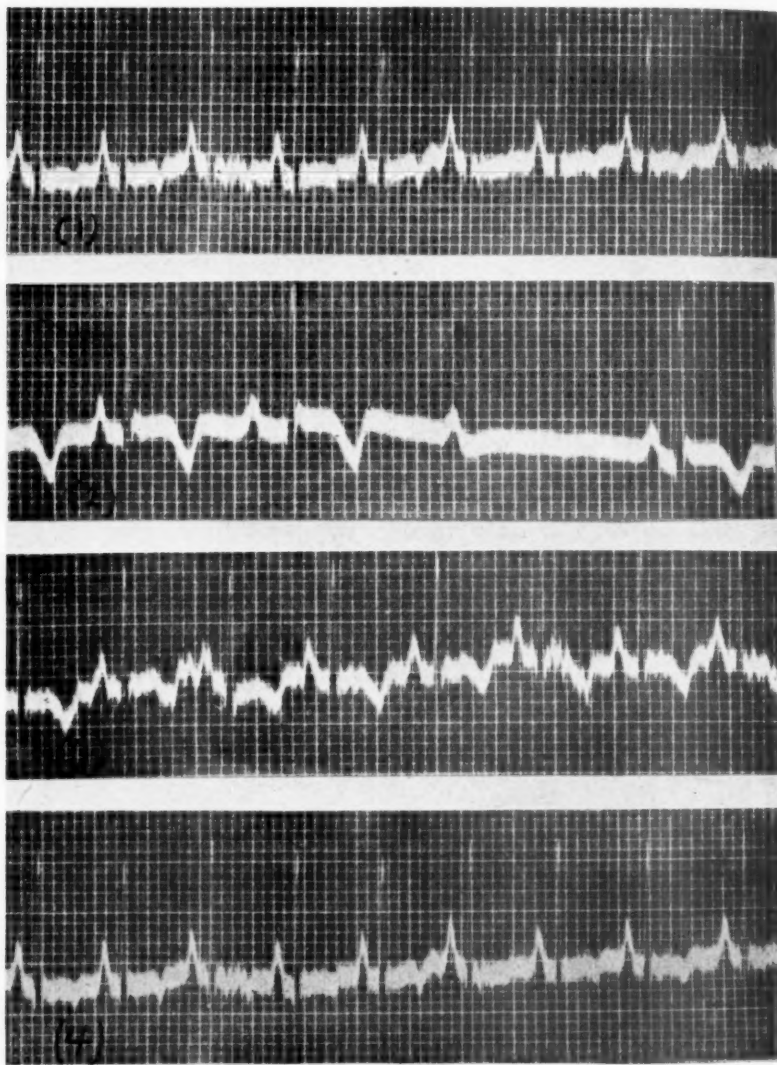


Fig. 2.—1, Control; 2, after 1 mg. morphine sulphate per kg. intravenously; 3, light ether anesthesia; 4, deep ether anesthesia.

This occurred in the absence of any signs of nausea or vomiting. It was followed by complete A-V block. Three minutes after the inhalation of ether was started the block was abolished, and the heart rate and P-R interval returned to the level present in the control period.

After a lapse of about three and one-half hours during which the animal recovered from the anesthesia, morphine effects upon the vagus were again in evidence. These effects were completely abolished after paralysis of the vagal endings by atropine. (See Fig. 4.)

In the fourth experiment a dog (B) received a dose of 20 mg. morphine sulphate per kg. subcutaneously. One hour later, during morphine narcosis, the tracing showed a very slow sinus rate (55 per minute) and long P-R interval (0.16 second). Three minutes after beginning the inhalation of ether (light anesthesia), the sinus rate had doubled, but the P-R interval, which ordinarily shortens with increase

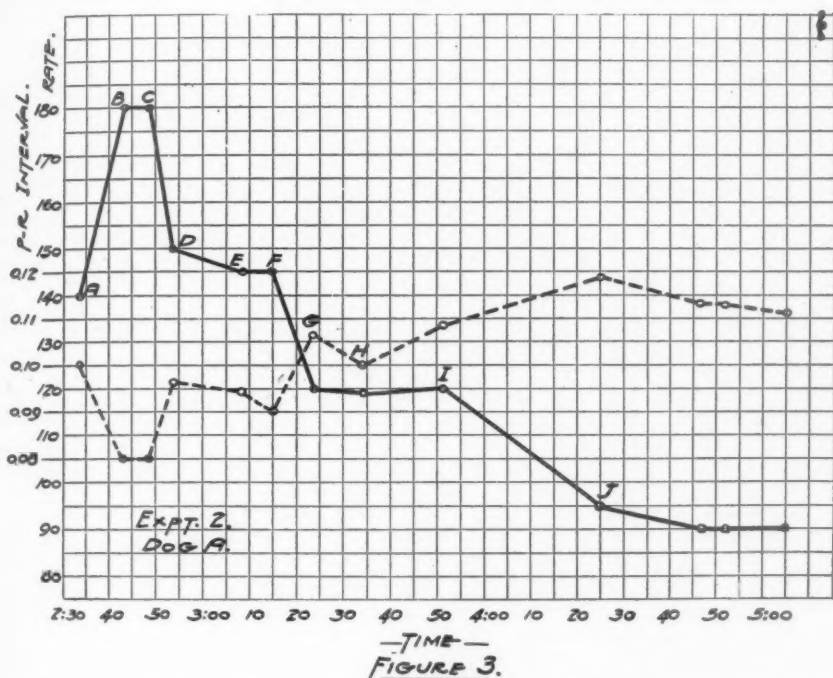


Fig. 3.—A, Control; B, nine minutes after starting inhalation of ether, corneal reflexes abolished; C, deep ether anesthesia; D, four minutes after 1 mg. morphine sulphate per kg. intravenously during deep ether anesthesia; E, and F, deep ether anesthesia; G, five minutes after 2 mg. morphine sulphate per kg. intravenously during deep ether anesthesia; H, deep ether anesthesia; I, seven minutes after ether discontinued; J, recovered from ether anesthesia.

in the rate, in this instance was lengthened to 0.24 second. During deep ether narcosis the sinus rate remained rapid and the P-R interval shortened to that present before the ether. In this experiment, therefore, the effect of a very large dose of morphine upon the sinus rate was abolished by the ether, while that upon conduction showed initial accentuation but at no time any diminution. Paralysis of the vagal endings by atropine produced further acceleration of the sinus rate (140 to 170) while the P-R interval diminished from 0.182 to 0.102 second. (See Fig. 5.)

In the fifth experiment an attempt was made to determine the rôle that stimulation of the accelerator nerves might play in the effects of ether. A dog (B_2) was given 10 mg. morphine sulphate per kg. intramuscularly and within fifteen minutes there was complete A-V block. Epinephrin, 0.023 mg. per kg. in a 1-10,000 solution injected intravenously, increased somewhat the rate of the ventricle, changed the shape of the idioventricular groups, but had no influence upon the A-V block. Ether inhalation promptly abolished the block and accelerated the sinus rate. A phenomenon similar to that seen in the fourth experiment occurred during the early period of ether inhalation—an increase in the sinus rate with further impairment of conduction.

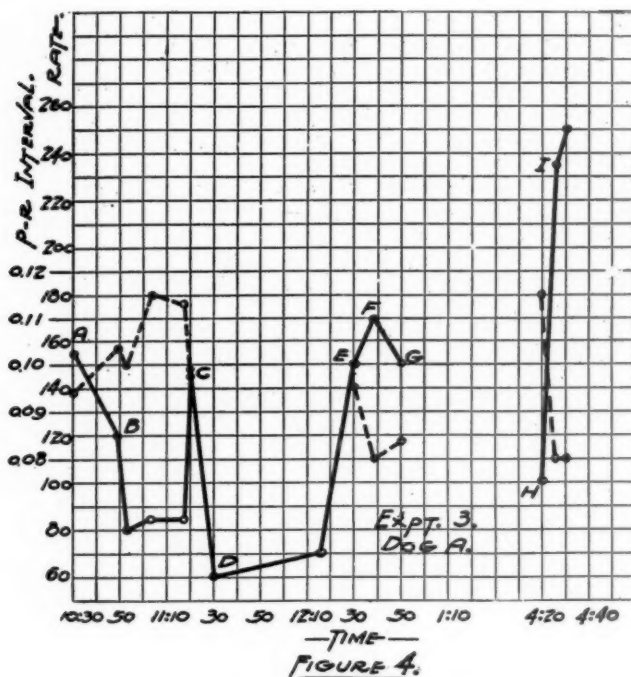


Fig. 4.—A, Control; B, one minute after 1 mg. morphine sulphate per kg. intravenously; C, two minutes after 2 mg. morphine sulphate per kg. intravenously; D, complete A-V dissociation; E, three minutes after starting inhalation of ether, light anesthesia, some struggling; F and G, deep ether anesthesia; H, completely recovered from ether anesthesia; I, one minute after 5 mg. atropine sulphate per kg. intravenously.

With the recovery from the ether, the vagal effects began to reappear. This experiment shows that the effects of the ether in all probability do not depend upon stimulation of the accelerators. (See Figs. 6 and 7.)

DISCUSSION

Ether is commonly employed for anesthesia in the study of the actions of drugs upon animals, and little attention is generally paid to

the possibility that these actions may be modified to an important degree by the anesthetic. Ether causes acceleration of the heart rate in the normal dog, and it has been shown that in the ordinary course of anesthetization by inhalation, it frequently produces changes in the cardiac mechanism seen in the electrocardiograms of animals¹ and man.² Eyster and Meek³ and Cohn⁴ observed that ether anesthesia frequently abolishes the arrhythmia produced by morphine. It has been reported that it diminishes the excitability of the vagus to elec-

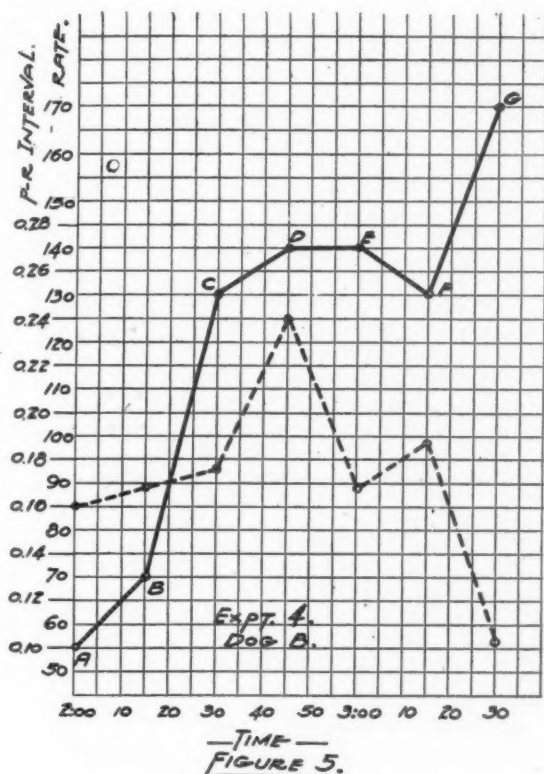


Fig. 5.—A and B, Controls, during morphine narcosis after 20 mg. morphine sulphate per kg. subcutaneously; C, three minutes after starting inhalation of ether; D, E and F deep ether anesthesia; G, two minutes after 0.3 mg. atropine sulphate per kg. intravenously.

trical stimulation. Thus Jackson and Ewing⁵ found that ether raises and morphine lowers the threshold for the reflex of cardio-inhibition produced by electrical stimulation of the divided left vagus. They did not show whether the effect was central or peripheral. Ruttgers⁶ found that after perfusing the frog's heart with 0.25 per cent ether, electrical stimulation of the vagus failed to produce slowing, and concluded that it paralyzes the endings of the vagus. There appears to be some evidence, however, that loss of response of the vagus mechan-

ism to electrical stimulation is not necessarily simultaneous with that to drugs. Witanowski⁷ observed that when the frog's heart is perfused with a solution of ether, response of the vagus and accelerator to electrical stimulation disappears while that to acetyl choline and epinephrin is retained for some period.

In the present series of experiments the effect of ether upon the susceptibility of the vagus to stimulation by morphine was studied. The results uniformly show that ether anesthesia interferes with the action of morphine on the vagus in the heart. Within limits, the effect of ether and morphine are mutually antagonistic. Thus the effect of small doses of morphine may be completely abolished by the induction of ether anesthesia as seen in Experiment I, and the acceleration

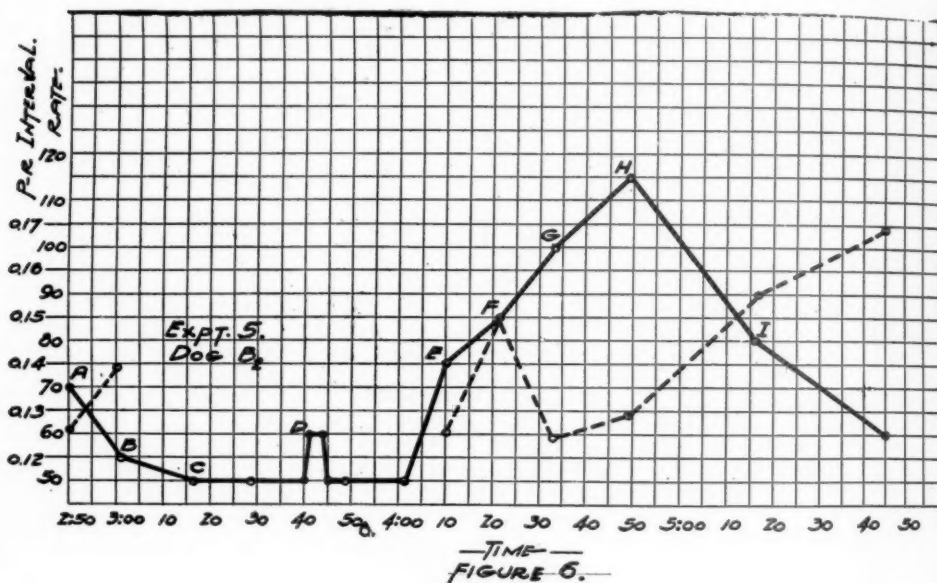


Fig. 6.—A and B, Controls; C, fifteen minutes after 10 mg. morphine sulphate per kg. intramuscularly, complete A-V dissociation; D, one minute after 0.023 mg. epinephrin 1-1000 per kg. intravenously; E, four minutes after starting inhalation of ether, slight struggling; F and G, deep ether anesthesia; H, fifteen minutes after ether discontinued; I, recovery from ether anesthesia.

of the heart during ether anesthesia may be overcome by a small dose of morphine as seen in Experiment II.

When ether inhalation was started, the animal often struggled violently, and it was thought that the effect attributed to ether may be due to stimulation of the accelerator nerves or reflex inhibition of the vagi as a result of the struggling. The fact that stimulation of the accelerators by epinephrin had no influence upon the morphine effect, whereas ether abolished the latter (see Experiment V) indicates that accelerator stimulation plays little, if any, part in the effect of the

ether, but that the latter effect is due to depression of the vagi. That it is not a reflex inhibition of the vagi, but in all probability a direct depression* of the center or endings by the anesthetic is apparent

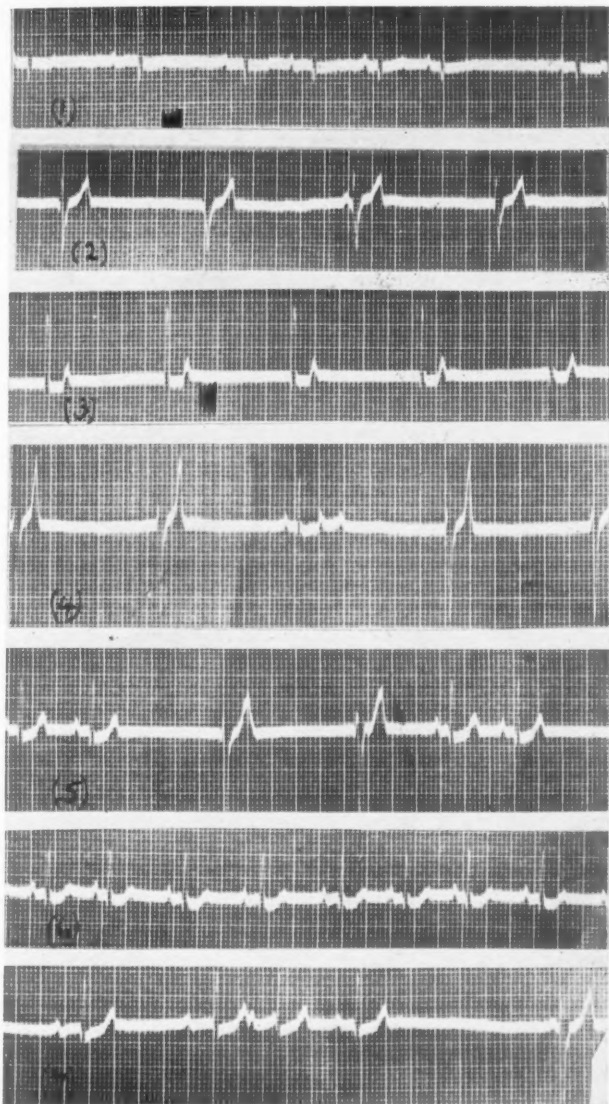


Fig. 7.—1, Control; 2, fifteen minutes after 10 mg. morphine sulphate per kg. intramuscularly; 3, two minutes after 0.023 mg. epinephrin per kg. intravenously; 4, four minutes after the epinephrin; 5, four minutes after starting inhalation of ether, light anesthesia; 6, deep ether anesthesia; 7, seventy-one minutes after ether discontinued.

from several experiments in which the animal, somewhat depressed by the morphine, did not struggle when ether was administered and

*Reflexes may contribute to the inhibition of the vagus, especially if the animal struggles.

a considerable quantity of ether was inhaled before there was any acceleration of the sinus rate or shortening of the P-R interval.

Ether depresses but does not paralyze the vagi in the heart in any doses that do not paralyze respiration. Atropine, after the full ether effects have appeared, causes further acceleration of the rate and shortening of the P-R interval.

The effects of morphine on the heart vary considerably in different animals. Cohn⁴ presented evidence indicating that some of these differences depend upon the fact that in one, the predominant effect is that of right vagus stimulation (marked sinus slowing); in another, the predominant effect is that of left vagus stimulation (marked disturbances in A-V conduction). On this basis it may be stated that ether usually depresses both nerves so that the rate is accelerated and conduction time is shortened. However, under given conditions, depression of the right vagus may be more in evidence than that of the left, as seen in Experiment IV, in which ether doubled the sinus rate but did not shorten the long P-R interval. This result probably depends upon the fact that after a large dose of morphine it requires much less ether to induce deep narcosis and the concentration of ether thus attained is insufficient to depress both vagi.

The initial lengthening of the P-R interval during ether anesthesia in this latter experiment, as also in Experiment V, is difficult to explain. That the sudden increase in the heart rate in the presence of impaired conduction might further depress conduction suggested itself as a possibility, but this explanation was refuted by the observation that after atropine, when the rate increased even further (from 140 to 170 a minute) the P-R interval shortened from 0.182 to 0.102 second. There are other possibilities, however, for which no evidence has been presented in this study, such as temporary local asphyxial changes affecting conduction, or that preceding the depression, there is brief stimulation of one vagus nerve, in evidence when its excitability has been greatly increased by a large dose of morphine.

SUMMARY AND CONCLUSIONS

1. Experiments were performed to study the effects of ether on the susceptibility of the cardio-inhibitory nerves to stimulation by morphine in the dog.
2. The results show that ether depresses the vagus and may abolish partially or completely the effects of stimulation of these nerves by morphine.
3. After large doses of morphine, deep ether narcosis may abolish vagus effects upon the sinus and not those upon conduction.

4. Ether by inhalation, in doses that do not paralyze respiration, depresses, but does not completely paralyze the vagi. Atropine is still effective after the full effects of ether have been induced.

5. Caution is necessary in the interpretation of the results of studies on the response of the vagus nerves in the dog during ether anesthesia.

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THE EFFECT OF OBSTRUCTION OF CORONARY ARTERIES UPON THE T-WAVE OF THE ELECTROCARDIOGRAM*

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THE T-wave of the electrocardiogram changes its form or direction when the myocardium is injured. This fact was observed experimentally by Eppinger and Rothberger¹ in 1909 and has had abundant confirmation in the human electrocardiogram. Eppinger and Rothberger further observed that the changes in the T-wave they induced tended to be opposite in character for the two ventricles. The injection of silver nitrate solution into the muscle of the right and basal portions of the heart caused the T-wave to become negative, whereas such injection into the left and apical portions of the heart resulted in a positive T-wave. They further found that spraying ethyl chloride on the heart also affected the T-wave, but the changes were opposite in direction to those following the injections. The effect of cold was confirmed by Wilson and Herrmann² and by Smith,³ and the experiments of Wilson and Finch⁴ indicate that the human heart responds in a similar manner.

I have previously shown⁵ that the ligation of the right coronary artery causes negativity of the T-wave while ligation of arteries supplying the left side of the heart has the reverse effect upon the T-wave of the electrocardiogram. Since these effects are similar to those observed by Eppinger and Rothberger,¹ they are probably due to similar causes, as it is functional impairment of a given area of muscle which produces the changes in the electrocardiogram when coronary arteries are closed.

A re-investigation of the effect of cold was made,⁶ and it was found that cooling the muscle, which prolongs the contraction, produced the effect reported by Eppinger and Rothberger, Wilson and Herrmann, and Smith; on the other hand, freezing the same areas of muscle so that their function was entirely abolished reversed the effect upon the T-wave. The facts suggest, therefore, the hypothesis that a tendency to negativity of the T-wave results from activity that is greater in duration or intensity in the muscle of the left lower portion of the heart, which may be actual or relative as a result of diminished activity in its antipode, the right upper portion of the heart. When the positive influence upon the T-wave is considered the circumstances are reversed.

Rothberger and Winterberg⁷ in 1910 observed that the activity of the accelerator nerves very markedly influences the T-wave of the elec-

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trocardiogram and that a distinct form is associated with the stimulation of each nerve. I⁸ have recently reported that the stimulation of the right accelerator nerve is often associated with a negative influence upon the T-wave, and the stimulation of the left accelerator nerve with a positive influence upon the T-wave—effects which are similar to those caused by injection of the muscle¹ or the closure of coronary arteries.⁵ If the above hypothesis is correct, accelerator nerve stimulation affects the T-wave of the electrocardiogram by decreasing the duration of the activity of the muscle affected.

Similar changes in the T-wave are also obtained by changes in the intracardiac pressure.⁹ A sudden increase in the intracardiac pressure of the right ventricle (clamping the pulmonary artery) causes temporary negativity of the T-wave and the same procedure in the case of left ventricle (clamping the aorta) tends to produce the reverse effect. Here the T-wave is affected perhaps because interference with the function of the muscle upon the side in which the intracardiac pressure is increased results in relatively greater function in the muscle of the antipode.

This report is concerned with the changes in the T-wave of the electrocardiogram when the three principal vessels of the heart (the right coronary artery, and the circumflex and anterior divisions of the left coronary artery) are obstructed. These vessels supply respectively the right upper and anterior, the left posterior and lower, and the intermediary or apical portions of the heart. The experiments, therefore, were actually determining the effect upon the electrocardiogram of interference with the function of large areas of muscle in these three portions of a heart.

The experiments were upon dogs under chloretone narcosis, with artificial respiration and the heart exposed by removing the sternum. The electrocardiograms were taken with the electrodes inserted into the right forepaw and left hind leg. There were eighteen experiments in all. In four, the mechanical movements of the heart were simultaneously registered from the base of the right ventricle after fixing the apex to the diaphragm with a single suture. In every instance the artery was obstructed by gently lifting it from its bed and applying a small clamp. The discoloration and loss of muscle function which follow¹⁰ occurred promptly. With the removal of the clamp the color and function of the muscle were rapidly restored and the electrocardiogram returned to the original form. The clamp was not applied longer than three minutes.

It has been pointed out by Lewis¹⁰ that the first changes in the rhythm of the heartbeat occur after a considerable interval following coronary obstruction. In these experiments there was no change in rhythm during the three-minute periods of occlusion.

The closure of the right coronary artery had a negative influence upon the T-wave of the electrocardiogram. The closure of the circumflex division of the left coronary artery had the opposite effect, namely, elevation of the T-wave. The closure of the anterior division of the left coronary artery tended to produce both effects, i.e., the T-wave began before the complete ascent of S, yet its peak became more positive. In two of the experiments the closure of this vessel caused only the positive effect upon the T-wave. These effects are shown in Fig. 1.

The zone of discoloration which followed the closure of the anterior division of the left coronary artery appeared at the lower portion of

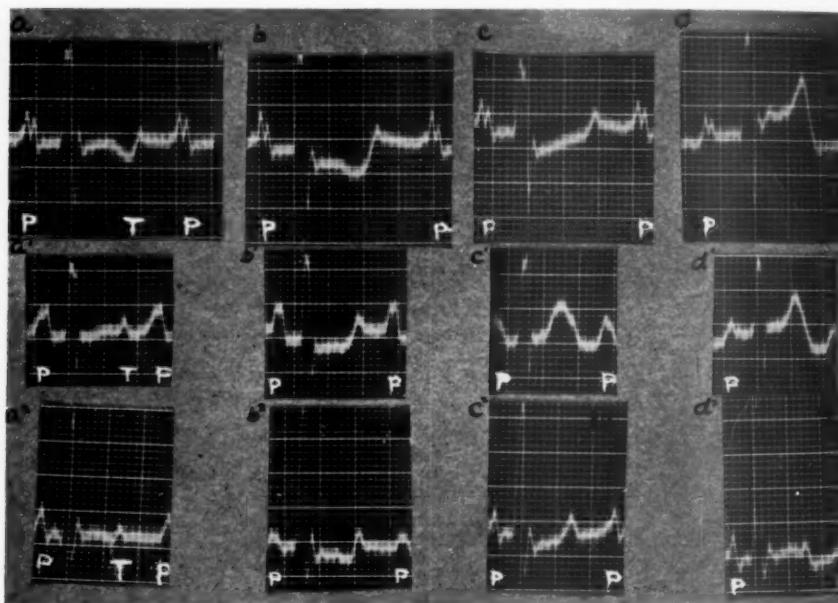


Fig. 1.—Axial electrocardiogram. Time in fiftieth seconds. *a*, The normal; *b*, closure of the right coronary artery; *c*, closure of the descending division of the left coronary artery; *d*, closure of the circumflex division of the left artery. *a*¹ *b*¹ *c*¹ *d*¹, The same, another experiment. In this animal only the positive influence on the T-wave from the closure of the descending division of the left artery appeared. *a*² *b*² *c*² *d*², The same, a third experiment. The small degree to which change occurred here was not due to an equivalent insignificance in the changes occurring in the muscle. The latter was as prominent as in other experiments.

the anterior interventricular groove and the apex of the heart. Dividing the ventricles into two halves, the one to the right above and anterior, the other to the left and posterior, it was, roughly, between the two. The involvement in the portion of the heart situated on its right side tended to cause negativity of the T-wave and involvement in the portion on its left side positivity of the T-wave. The expectation, therefore, that the closure of the anterior division of the left coronary artery would produce less characteristic change in the form

of the T-wave than would be the case with the other two vessels, and that the change which occurred would partake of the nature of that associated with the closure of one or both of the other two was fulfilled.

In three of the experiments, immediately after the closure of the circumflex division of the left artery, the T-wave became negative before the usual effect, positivity of the T-wave, appeared (Fig. 2). This fleeting reversal of the effect upon the T-wave is similar to that which is often observed when the accelerator nerves are stimulated.⁸

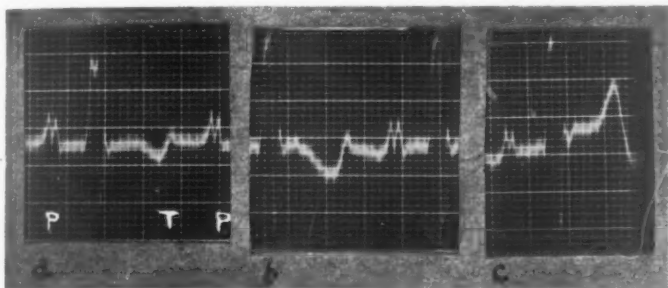


Fig. 2.—Axial electrocardiogram. Time in fiftieth seconds. *a*, Normal; *b*, the earlier effect of the closure of the circumflex divisions of the left coronary artery, which passed quickly into *c*, the enduring effect.

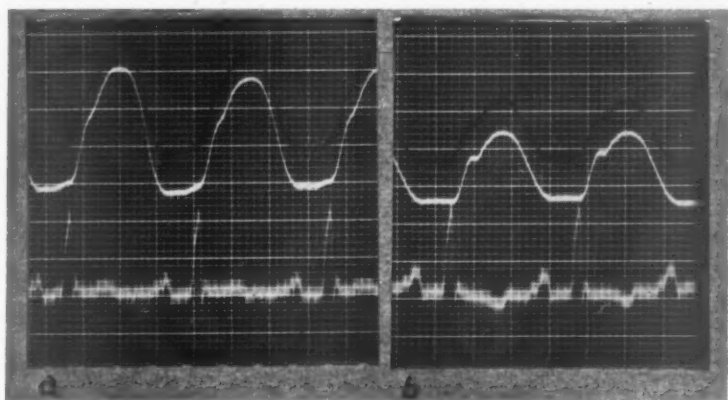


Fig. 3.—Axial electrocardiogram with mechanocardiogram. *a*, Normal; *b*, closure of the right coronary artery. The electrocardiogram presented little change, although the mechanocardiogram indicated there was marked diminution in the activity of the right ventricle.

There was no relation between the degree to which the T-wave was altered and the relative size of the area of the myocardium involved. With extensive involvement of the myocardium the resulting electrocardiographic changes may be insignificant. In Fig. 3 the effect upon the mechanocardiogram and the electrocardiogram is seen after the right coronary artery was clamped. Closing the lumen of this vessel

impairs the function of a large portion of the outer wall of the right ventricle. This experiment presented an unusual contrast between the two because the change in the T-wave was insignificant.

SUMMARY AND DISCUSSION

The experimental occlusion of the principal coronary arteries produces changes in the T-wave of the electrocardiogram which vary in direction depending upon the vessel involved. When the muscle of the right and anterior portions of the heart (including the basal portion in that region) is involved by the occlusion, the T-wave tends to become negative, and when the muscle of the left and posterior portions of the heart is involved, the reverse is the case; the T-wave tends to become positive. These and other observations cited suggest that the T-wave of the electrocardiogram of the intact heart *in situ** reflects the disturbances which occur in the balance of the electrical activity of the two halves of the heart (the halves correspond approximately to the right and left ventricles). When a change in the heart muscle occurs, the effect that is exerted upon the T-wave depends upon the location of the injury with respect to the two halves of the heart.

The axial lead was employed for these experiments. That the line of the leading also plays an important rôle in the form of the T-wave is suggested by the work of Katz and Weinman.¹¹ These authors concluded, however, that the T-wave is due to an unequal duration of the activity of the various fractions of the ventricles. Further evidence is visible in the fact that the curves of simple strips of heart muscle,^{12, 13} electrograms, do not essentially differ from curves of the entire heart, electrocardiograms. I have called attention¹⁴ to the influence that the line of the lead may have in determining the form of the T-wave after the stimulation of the accelerator nerves.

Further investigations along these lines will be reported in subsequent communications.

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*The view is not concerned with the factors which cause the changes in the T-wave of the electrogram.

MYXEDEMA HEART*

WITH REPORT OF TWO CASES

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THE so-called myxedema heart was first recognized by Zondek¹ in 1918. As characteristic features he described the marked enlargement of the left and right ventricles of the heart, slow pulse rate, normal blood pressure, and electrocardiographic changes.

In his first paper, Zondek reported four cases of myxedema which showed the classical features of the disease, and in which the tele-roentgenograms revealed an enlargement of the heart both to the right and to the left, with a marked reduction in its transverse diameter after thyroid therapy. The electrocardiograms of his patients showed low or absent P-waves, absent T-waves, and changes in the QRS complexes. The intraventricular conduction defects disappeared with the reduction in the size of the heart when thyroid extract was administered. As these patients improved, the basal metabolic rates increased.

The transverse cardiac diameter in Zondek's first case measured 19.7 cm. In order to test whether this "high grade dilatation is in reality an expression of the myxedematous syndrome," he studied the influence of thyroid substance on the size of the heart. After four weeks of this therapy, there was a definite reduction in size; the transverse diameter now measured 17.8 cm. Eight weeks after the beginning of treatment, there was a still more marked reduction, to 14 cm., which was the normal for that individual patient. The changes noted on fluoroscopy were that the heart appeared as a "lifeless, expressionless mass with deformed contour"; "a livelier action and normal cardiac silhouette were revealed" after therapy. The electrocardiograms also returned to normal. Corresponding changes were obtained in the hearts of the three other patients. The illustrations of Zondek's roentgenograms and electrocardiograms before and after treatment are very striking. Zondek thought that the marked reduction of the size of the myxedema heart suggested a specific effect, but the details of his theory will be discussed later in this paper.

Since his original observations, there have been few contributions to the subject of myxedema heart. Assmann² in 1919 reported a case confirming the findings of Zondek. Assmann's patient had a pulse rate of 50 beats per minute, a transverse cardiac diameter of 16.7 cm.; but following thyroidin, 0.3 gram daily for three weeks, the

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transverse diameter was reduced to 12.7 cm., and the pulse rate increased to between 70 and 80.

In his 1918 paper, Zondek showed that the myxedema heart resulted from the damage to the myocardium and not to the nervous elements of the heart and that the therapeutic influence of thyroid extract is on the muscle. In his second paper, published in 1919,³ he reported two further cases. One, a sixty-two-year-old woman, showed mild signs of cardiac insufficiency and had a blood pressure of 160 mm. and a pulse rate of 52. The x-ray plate showed enormous dilatation of all the cardiac chambers and marked widening of the aorta. It is of particular interest to note that the electrocardiogram showed absent P- and T-waves. In addition, there were ventricular extrasystoles in which the T-waves were present. This brought up the question whether the muscle of the myxedema heart was in a state of diminished susceptibility to excitation or was of diminished contractile power. This was suggested because the extrasystolic contraction revealed the fact that the myxedema heart is capable of eliciting a T'-wave. It is not known whether the normal T-wave and the T'-wave of an extrasystole are due to one and the same phenomenon. His second case, a forty-two-year-old major, has frequently been quoted because of the unusual development of the disease in this patient. This man received a gun-shot wound in his neck which resulted in a long period of supuration of the thyroid gland. He then developed typical signs of myxedema and cardiac insufficiency due to the presence of a myxedema heart, the symptoms of which began twelve months after the original injury. These findings were confirmed by x-ray films and electrocardiograms. The latter showed absent P- and T-waves. After three weeks of thyroid treatment, the symptoms of cardiac insufficiency had subsided and the transverse cardiac diameter had been reduced by 1 cm.

Meissner, in 1920,⁴ reported three similar cases. The first was a patient of fifty-six years in whom there was marked cardiac enlargement to the left and right; the sounds were rough, the second apical sound was not accentuated. The pulse rate was 50. The teleroentgenogram showed a transverse diameter of 20 cm.; after treatment the measurement was reduced to 15.5 cm. A comparison of the findings in the x-ray plates before and after thyroïdin is very striking, especially when one considers that myxedema had been present for twelve years. The second patient had a blood pressure of 180 mm. and a pulse rate of 58. Examination of the heart, blood pressure, and urine suggested a chronic nephritis, but the entire picture was decidedly in favor of myxedema. After two months administration of thyroid, there was a marked improvement of the patient's condition, the pulse rate now being 66 and the blood pressure 160 mm. systolic. It is very interesting, however, to note that the x-ray plate in this case showed no change in the size of the heart. The author interprets this lack

of reduction as due to a complicating chronic nephritis. This, he states, may explain the failure of the influence of thyroid substance on the size of the heart. The third case showed a normal heart before thyroid extract was administered and no change in the cardiovascular system afterwards. Thus only the first case in this report resembles the typical ones of Zondek.

It was not until 1925 that the first paper dealing directly with myxedema heart appeared in this country. This was a communication by George Fahr⁵ who emphasized the rarity of the condition and stated that he was able to collect only eight cases in the literature up to that time. He further stated that he had seen three cases in one year. There has been a moderate amount written in the American literature concerning observations on the heart in myxedema, but very few authors have published x-ray and electrocardiographic reports.

Fahr's first case was typical myxedema in a woman of forty-six years who had all the signs of cardiac insufficiency. The pulse was 70, blood pressure 110/70 mm.; and the basal metabolic rate was minus 25 per cent. The teleroentgenogram showed right and left cardiac enlargement, with a transverse diameter of 17 cm. This patient was given rest in bed and tincture of digitalis for three weeks. Under this régime there was very little improvement in the edema, dyspnea, and other cardiac complaints. Digitalis was then stopped and eight grains of thyroid extract were administered each day. A marked improvement in the cardiac condition resulted, so that seven weeks later the transverse cardiac diameter was 14.5 cm. At this time the basal metabolism was plus 8 per cent, the pulse rate 80, and the blood pressure 120/80 mm. This author controlled his results by a therapeutic test. He discontinued the thyroid medication, and there was a return of all the signs of myxedema, including those of cardiac insufficiency. The metabolism rate diminished and the teleroentgenogram proved that the heart had again enlarged, its diameter was then 15 cm. Medication with thyroid extract was resumed, and six weeks later the signs of myxedema again disappeared, the basal metabolic rate increased, and the heart became normal in size. Parallel findings were noted in the electrocardiograms. On admission there was an intraventricular block with negative T-waves in Leads I and II; the ventricular complexes were negative in Lead III, and the P-waves were normal. After thyroid medication was tried, the electrocardiograms became normal, but when the medication was discontinued, the characteristic intraventricular conduction defects reappeared, with negative T-waves in Leads I and II. Fahr's second case showed mild symptoms of cardiac failure, with typical x-ray and electrocardiographic changes of myxedema heart, which returned to normal after treatment.

A further review of the American literature will be given following these two references to the French.

Laubry, Mussio-Fournier and Walser,⁶ in 1924, reported a case of myxedema in a patient who complained of typical anginal attacks and who showed cardiac hypertrophy without valvular lesions. Digitalis and other remedies gave no relief, but when thyroid medication was tried, there was marked clinical improvement, with a corresponding reduction in the cardiac volume.

P. Abrami and his coworkers⁷ reported two cases, in both of which the patients complained of precordial pain. These authors do not present a very careful, detailed study of their cases and do not report the electrocardiograms. One is not convinced that they were actually dealing with myxedema heart, but one gets the impression that their cases were probably instances of coronary thrombosis.

Christian in 1925⁸ stated that although he had seen thirty-two cases of myxedema at the Peter Bent Brigham Hospital, he had not observed one case like those described by Fahr, and suggests the rarity of the myxedema heart. Sturgis,⁹ in his paper entitled: "The Treatment and Prognosis in Myxedema," a study based upon twenty-six patients, refers to Fahr's article and states that in this disease, serious impairment of the myocardium is amenable to thyroid medication. He discusses at length the therapy in these cases, but he does not report any definite cases of myxedema heart which resemble those of Zondek, Assmann, or Fahr. This same writer in 1926¹⁰ reported a case in a woman of fifty years, who had myxedema and who also complained of anginal attacks. The basal metabolic rate of this patient was minus 32 per cent and her blood pressure was 135/110 mm. On clinical examination the left border of the heart was 9.5 cm., and the right border 3.5 cm., from the midsternal line. He did not mention the electrocardiogram or x-ray findings. In another case of myxedema in a man of fifty-seven, who complained of dyspnea and substernal oppression, the basal metabolism was minus 37 per cent. The tele-roentgenogram showed "uniform and rather striking enlargement of all four chambers of the heart." Thyroid extract was administered, but the patient still complained of substernal pain on slight exertion. No note was made concerning the x-ray result after treatment, nor did he mention any reduction in the size of the heart. Electrocardiographic reports were also omitted. It is possible that the cardiac enlargement in this case may have been due to a hypertension and not to a primary dilatation. Sturgis again wrote on the cardiovascular system in myxedema in 1927.¹¹

Willius and Haines¹² studied 162 cases of myxedema with particular reference to the cardiovascular system. Eleven cases, or 7 per cent, showed evidences of cardiovascular damage, such as cardiac hypertrophy and myocardial insufficiency associated with hyperten-

sion, renal disease, arteriosclerosis, or angina pectoris. In one man of forty-eight years, who complained of dyspnea and extreme weakness, a moderate cardiac hypertrophy with bradycardia was found. The electrocardiogram gave evidence of prolongation and notching of the QRS complexes in all leads. These findings disappeared after thyroid extract was given. These authors found no change in the size of the heart, but they did not publish the teleroentgenograms. "Subsequently, the T-waves in Lead II and III became negative, increasing the probability of the associated degenerative myocardial changes." Here, again, it is questionable whether this case is a true myxedema heart. Electrocardiographic studies were made in 55 of their cases; 28 of which were found normal; in 12 the results were incomplete; in 12 others the abnormalities disappeared after thyroid administration. From their data they conclude that a cardiac syndrome of myxedema does not exist.

Means, White, and Krantz¹³ collected 48 cases from the Massachusetts General Hospital where they looked particularly for the cardiac changes in myxedema. They report one case in which the cardiac enlargement subsided when thyroid substance was given. This was a woman of forty-four years, a typical myxedema patient, in whom the basal metabolism rate was minus 34 per cent, the pulse rate 66, and the blood pressure 148/90 mm. The electrocardiogram was normal. The seven foot plate of the heart revealed a general enlargement, with a transverse diameter of 14 cm. This patient was given thyroid extract and fifteen days later, the basal metabolic rate rose to minus 4 per cent and the x-ray plate showed a transverse cardiac measurement of 11.7 cm. The pulse rate rose to 100 per minute. Incidentally, this patient died suddenly sixteen days later. These authors believe that the condition of myxedema heart exists, but that it is exceedingly rare.

Curschmann, in 1926,¹⁴ reported a case of myxedema heart in a man sixty years old, with results similar to those of Zondek and Meissner. In a woman of forty-seven with myxedema heart and cardiac insufficiency, Zins and Rösler¹⁵ showed a reduction of the transverse cardiac diameter from 14.9 cm. to 11.6 cm. after three weeks of thyroïdin.

Another case report was published recently by Schittenhelm and Eisler.¹⁶ The basal metabolism rate was minus 20 per cent, the blood pressure 150/96 mm., and the pulse rate between 60 and 70. They reproduced the x-ray plate, which demonstrated a marked cardiac enlargement to the right and left, the transverse diameter measuring 18 cm. The electrocardiogram showed a small P-wave, negative T-wave, and small waves in the first and second leads. They administered 12 mg. of thyroxin intravenously in eleven days. They then demonstrated a reduction in the transverse cardiac diameter to 14.7 cm. and a normal electrocardiogram.

In 1927 Fahr¹⁷ reported six cases of myxedema heart including the first case reported in his earlier paper.

Because of the scarcity of case histories in the study of myxedema heart, the following two cases of myxedema, with the typical roentgenographic findings, seem of sufficient interest to report. The first patient was studied on the medical service of Doctor B. S. Oppenheimer, to whom I am greatly indebted for the privilege of reporting the findings.



Fig. 1.—Appearance of the patient, L. S. (Case 1), before thyroid extract was given. Note the expressionless face, the thick lips, and the pigmented dorsal surface of the hands.

CASE 1.—L. S., aged fifty-eight years, who had been married for thirty years, had four living children. He was known to have had myxedema for eighteen years, but he had never adhered strictly to treatment and had never been studied in any one clinic. He showed the characteristic slow mental reactions and other features of the disease. When first seen, he took very little interest in his environment, was very uncooperative and refused to answer direct questions. Typical answers in his history are: "I don't care," and "What difference does it make?" He was once a painter, but had not worked for years because he felt "sick and weak." He always complained of feeling cold, seldom shaved, and had not had his hair cut

in two years. Examination revealed an expressionless face, thick lips, diminished palpebral fissures, pads of thick tissue about the eyes and supraclavicular swellings. He passed most of the days in sleep and seldom left his bed. Other classical features, such as dry skin, irregular borders of the hair distribution, and slow, sluggish speech were noted. There was an absence of axillary hair and scanty pubic hair. The tongue was thick and pudgy. The thyroid gland was not palpable. Examination of the heart showed that it was enlarged. The pulses were equal, regular, of fair tension, and rate of about 66 per minute. On palpation no demonstrable thickening of the peripheral vessels could be detected. The blood pressure was 110/68 mm. There was dullness over the right lower lobe of the lung and a few crepitant râles were heard. The hands and feet were short, and the skin was thickened. The scrotum showed patches of vitiligo. The dorsal surfaces of the hands revealed a roughened, scaly, brown pigmented skin. Examination of the blood revealed a moderate secondary anemia. The basal metabolic rate before treatment was minus 20 per cent. The urine contained no albumin

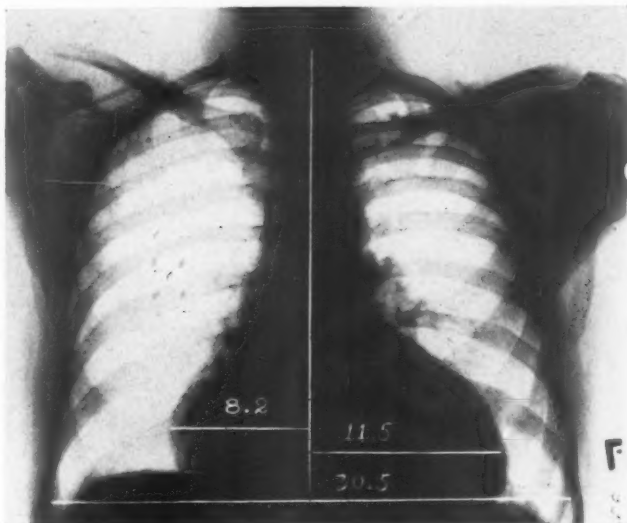


Fig. 2.—This teleroentgenogram shows the dilatation of the heart involving chiefly the right and left ventricles. The transverse diameter measures 19.7 cm. This plate was taken shortly after his hospital admission. The pneumonic process at both bases is not well reproduced.

or casts; the phenolsulphonephthalein output was 65 per cent in two hours; and the kidney showed good concentrating power. The blood chemistry was as follows: urea nitrogen 18 mg., cholesterol 258 mg., calcium 9.6 mg., and blood sugar 98 mg. He refused to have a Janney test performed. The blood Wassermann was negative. He weighed 152 pounds. The temperature was usually around 98 degrees F. Fig. 1 shows the appearance of the patient during the first few days of his admission to the ward.

Before ordering thyroid medication, the teleroentgenogram was taken. It showed (Fig. 2) a moderate dilatation of the left ventricle and a marked enlargement of the heart to the right. There were the remains of a pneumonic process at both bases of the lungs. The electrocardiogram taken at this time showed low voltage in all three leads, a left ventricular preponderance, notching and prolongation of the QRS complexes in all leads (their interval was prolonged to 0.12 seconds), and flattening of the T-waves in Lead III (Fig. 3). The patient was

put on thyroid extract, six grains per day, and shortly afterward, he showed the usual dramatic improvement. He became more alert; began to feel warm for the first time in years, and asked to get out of bed. The basal metabolism rate gradually rose, so that on the tenth day it was minus 12 per cent. An x-ray plate taken three weeks later showed a disappearance of the inflammatory process at the bases but no change in the size of the heart. Thyroid extract was continued until a total of 173 grains had been given in seven weeks. At this time the patient's pulse became more rapid; there was a marked improvement in his mental attitude and alertness. He began to take an interest in his person, and shaved twice a week. His blood pressure was 108/64 mm. He felt so much bet-

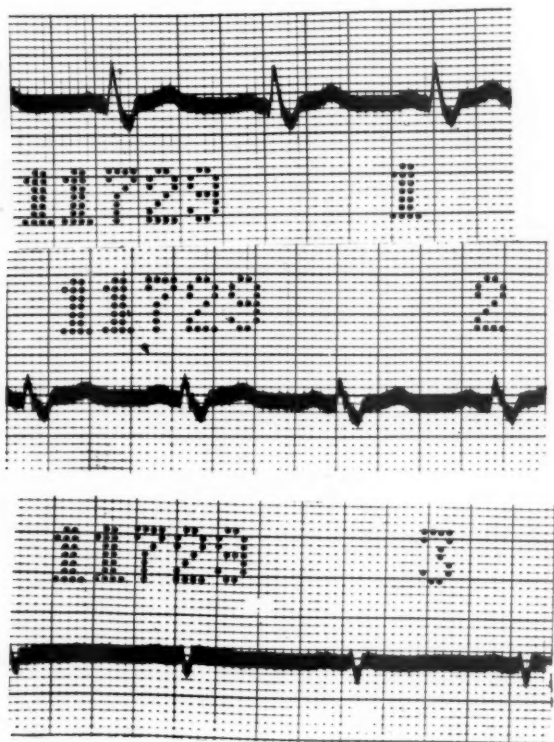


Fig. 3.—Three leads of the first electrocardiogram before treatment are shown. See text for description.

ter that he was discharged. At this time his basal metabolism was plus 9 per cent. Another x-ray was taken which revealed no change in the size of the heart. Another electrocardiogram showed even better than the previous one the notching of the QRS in all three leads, the low voltage in all leads, the inversion of the T-wave in Lead III, and an intraventricular conduction defect.

After discharge the patient was given maintenance doses of thyroid extract and was seen at intervals of two weeks. He claimed to have adhered to his therapy regularly. There was such a marked improvement that he said his own friends did not recognize him. Two months later a seven-foot plate of his heart showed no demonstrable reduction in size (Fig. 4). The apparent difference in the transverse diameter could well be explained, according to the interpretation of Dr. Harry Wessler, as resulting from changes in the height of the diaphragm which

occur during the respiratory cycle. Variations in the technic of taking the plates may account for slight changes heretofore reported in the literature. His basal metabolic rate now was minus 4 per cent, his pulse rate 70. He did not complain of any feelings of discomfort. An electrocardiogram taken two months after treatment also revealed no definite changes as compared with those taken earlier.

One of the most striking features in this case is the absence of reduction in the size of the heart and of improvement in the electrocardiographic changes, despite thyroid administration which was otherwise efficacious. There is a marked contrast between those cases of myxedema in which the electrocardiographic and roentgenological abnormalities disappear after thyroid medication (Zondek, Assmann, and Fahr) and those, like the case just cited, in which no definite

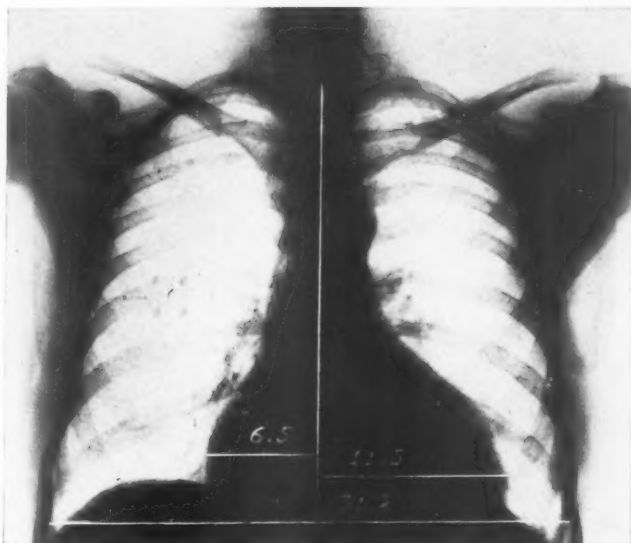


Fig. 4.—The result after two months of thyroid therapy. Although there is an apparent reduction in the transverse cardiac diameter (18 cm.), this may be due to differences in the height of the diaphragm.

changes could be demonstrated. One may conclude that in myxedema the cardiac manifestations fall into two groups: In one are those cases in which the administration of thyroid extract results in reduction of the cardiac size and a disappearance of the abnormalities in the electrocardiogram. In the second group one may consider those cases of myxedema in which the roentgenological and electrocardiographic evidences point to cardiac changes, but wherein one can find no amelioration after a sufficient course of thyroid extract.

In a personal communication to the author, Zondek stated that he has seen two such cases as the one reported in this paper, in which the cardiac manifestations did not return to normal after thyroid therapy. This suggests a definite organic myocardial change in the

latter group, not amenable to the influence of thyroid medication. Case 1 resembles the second case of Meissner, already quoted, where similar observations were made. It is possible that the long duration of myxedema (over eighteen years) may have been a factor in the production of organic changes in the heart; changes of such a character that they could not respond to thyroid therapy. These observations may be of further help in establishing the mechanism of the reduction in the size of myxedema heart in such cases as those quoted by Zondek. He stated that the apparent enlargement of the myxedema heart was due to a dilatation, because of the rapid reduction in size in response to thyroid. The characteristic cases in the literature show that when reduction occurred, it usually took place within a few weeks. The

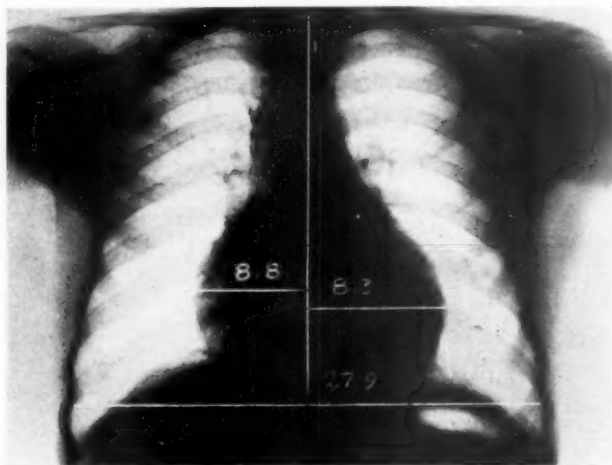


Fig. 5.—Teleroentgenogram of Case 2 (M. H.). There was a general enlargement of the cardiac shadow to the right and left.

case presented in this report was observed for over four months. No further changes in the size of the heart may be expected. This we interpret as an expression of cardiac hypertrophy *per se* and would expect to find, histologically, fibrosis of the myocardium.

CASE 2.—M. R., twenty-six years old, male, single, was studied in the dispensary service of Dr. Harry Wessler. This patient had always been a "good student," but was very phlegmatic and could never hold any responsible position. He complained of "inability to work." On examination, the puffiness of his eyelids and the thick, broad tongue were prominent features. On percussion the heart was enlarged to the left. There was a moderate pretibial edema, a palpable liver, and an enlarged spleen. The nonprotein nitrogen was 35 mg., the blood cholesterol 200 mg. The total protein and differential blood picture were normal. The blood volume was found to be 3.1 or 50 c.c. per kilo. The initial basal metabolic rate was minus 18 per cent. The patient weighed 139 pounds, his blood pressure was 100/60 mm., urine was negative.

X-ray films of the skull were negative. A teleroentgenogram (Fig. 5) showed a "marked enlargement" of the heart. "It is globular in shape and under the fluoroscope the cardiac contractions were seen to be very poor." The electrocardiogram revealed a tendency toward a left ventricular preponderance.

ELECTROCARDIOGRAPHIC CHANGES

There have been many electrocardiographic studies in thyroid insufficiency. Some of the findings have already been quoted. Zondek¹ originally observed low P- and T-waves. He thought that electrocardiograms were the best guides to therapy and suggested their use in control.¹⁸ It is difficult to explain why the P- and T-waves are absent and return after treatment.

Zondek¹⁹ stated that there is a different underlying condition to explain the diminished action and power of the myxedema heart from that in myocardial insufficiency due to other causes. He thinks that the return of the T-waves after treatment is due to a gradually increasing strength of the heart's action. By means of phlebograms^{19, 20} he demonstrated that the auricular wave is absent in this disease. The auricle, however, is not "still"; a number of minute twitchings are shown, which may be due to an "auricular tachycardia." The dilatation of the auricle is passive, the so-called myogenic dilatation due to stasis. There is a diminution, consequently, in the propulsive force of the heart. Thus he explains the absence of P-wave. The subsequent work of Lueg²⁵ and of Nobel, Rosenblüth and Samet²³ offer a better explanation.

Thacher²¹ studied electrocardiograms of 8 cretins and concluded that the T-wave is flattened, low, or even inverted in cretinism and returns to the normal after thyroid administration.

Gardner²² reported a case in which there was a splitting and widening of the QRS complex and negative T-wave in Lead I with a return to normal after giving thyroid extract. Nobel, Rosenblüth, and Samet²³ also studied the electrocardiograms in thyroid insufficiency and came to similar conclusions. Thacher and White²⁴ showed in every one of 14 cases of myxedema, a decrease in the height of the T-wave in Lead I as compared with the normal. No auriculo-ventricular or intraventricular block was noted. The P-R interval was usually normal. The QRS complexes in some cases showed a decreased amplitude and "increased as did the T-wave with thyroid." These authors draw a definite relation between the T-wave as observed in the electrocardiogram and the basal metabolic rate in myxedema.

Fahr does not agree with Zondek that the P-wave is necessarily absent in the electrocardiograms of the myxedema heart, because it was practically normal in his cases. He is of the opinion that a negative T-wave in Leads I and II is the most characteristic change in the electrocardiogram and that the QRS changes are less common. In his

first case, the first change to normal after the administration of thyroid extract was in the T-wave in Lead I, which was diphasic at first and later positive. Next the slurring of the QRS became normal and the conduction time decreased.

Lueg²⁵ attempted to explain the electrocardiographic changes in hypothyroidism by an altered electrical capacity of the skin rather than by myocardial changes.

Nobel, Rosenblüth and Samet state that the increased resistance of the skin and tissues could, in part, account for the changes in the electrocardiograms. To decide this question they investigated the skin resistance, which was known to be increased, and then attempted to reduce it. In place of the usual plate electrodes they substituted needle electrodes, both on the extremities and thorax. The resulting electrocardiograms taken by the new technic (employing one needle electrode in the sternal end of the second right intercostal space and the second needle in the region of the cardiac apex) revealed the presence of P- and T-waves. They concluded that there are no changes in the conductive mechanism of the myxedema heart, but that the electrocardiographic changes are expressions of the abnormal skin resistance and, therefore, an indirect evidence of metabolic changes. These conclusions speak against the theory of Zondek; namely, that the absence of P- and T-waves is due to a disordered cardiac conduction mechanism.

THYROID MEDICATION

Sturgis and Whiting⁹ administer thyroid gland, 0.13 gm., three times a day for five days. The patient is carefully watched for such untoward signs as loss in weight, rapid pulse rate, and complaints of palpitation, dyspnea, dizziness, nausea, or a feeling of warmth. If these do not appear, they continue giving thyroid tablets, 0.13 gm., twice a day, continuing until the pulse is about 75. Maintenance doses of 0.13 gm. daily are given when a satisfactory basal metabolic rate has been reached.

These authors advise a careful watch for untoward signs, because some patients with myxedema have serious cardiac impairment. They report the case of a woman of fifty years (quoted above¹⁰) who died suddenly during her treatment. At post-mortem examination, coronary arteriosclerosis, cardiac infarction, and hypertrophy with dilatation were found. Christian sounds the same warning. These authors suggest that after thyroid treatment, the increase in the metabolism throws an added burden on an already damaged myocardium which is unable to meet the strain.

Zondek also warns against treating patients with thyroid extract without sufficient grounds. He thinks that in the absence of cardiac

enlargement and electrocardiographic evidences, care should be exercised in the use of thyroid extract.

Swan²⁶ reports a very interesting case of myxedema in which auricular fibrillation appeared when thyroid substance was given, ceased when thyroid was discontinued, but reappeared when the drug was readministered. Again the therapy was stopped and the rhythm became regular. In his case the electrocardiogram showed a prolongation of the P-R interval (0.24 seconds).

CARDIAC PATHOLOGY IN MYXEDEMA

Little is known concerning the pathological findings in the heart in myxedema. After extirpation of the thyroid gland, Kish²⁷ and Bensen²⁸ have found degenerative changes in the myocardium, describing first the loss of transverse striations with granular disintegration; and, later, cloudiness of the muscle fibers. C. Wegelin²⁹ in an exhaustive study of the thyroid states that the most common anatomical finding was the dilatation of the heart.

Goldberg³⁰ performed thyroidectomies in sheep and goats. In 11 out of 17 sheep he found calcification of the aorta, cardiac dilatation and a degree of atherosclerosis not found in his control animals. He described a flabby myocardium in which microscopically no cross striations could be seen. The fibers were found to be densely packed with deeply staining nuclei. There was also vacuolation of the Purkinje fibers. A common post-mortem finding was calcified plaques in the pulmonary artery and in the thoracic and abdominal aorta.

Schultz³¹ found a peculiar infiltration of mucoid substance and a thickening of the aortic valves. He also found a disintegration of the muscle fibers.

Other observers have noted the sclerotic processes in the aorta and coronary arteries. A. Fishberg³² pointed out the frequency of atherosclerotic changes in myxedematous patients involving commonly the kidneys, and frequently associated with hypertension and myocardial insufficiency. In discussing the myxedema heart, he suggests that arteriosclerotic changes may indirectly play a rôle in the pathogenesis of this condition. These observations have been frequently made in myxedema. It will be recalled that in Meissner's second case (see above) there was an associated hypertension and chronic nephritis. These findings may well explain the failure in the reduction of the cardiac volume after thyroid administration in some cases.

In an experimental study of the morphology of the heart muscle in hypothyroidism, Brooks and Larkin³³ performed thyroidectomies on rabbits, the animals being examined after death, which varied from two to forty-three days postoperatively. These authors conclude that

cardiac signs of myxedema are not due to alterations in the myocardium because no characteristic pathological picture could be detected in their studies.

MECHANISM OF THESE CHANGES

The last authors quoted emphasized the observations that the cardiac signs in myxedema appear quite early. They do not believe that these changes are of an anatomical character, the relief following thyroid treatment being against this theory. Means and his collaborators consider the cardiac changes to be functional, directly due to thyroid insufficiency as proved by the relatively rapid response to thyroid therapy. They differentiate this type of case from those myxedematous hearts in which there are organic changes.

Zondek, in his first paper, raised the question regarding the mechanism in the cardiac reduction in this disease. He states that there is dilatation of the heart and not hypertrophy. He reasons as follows:³⁴ the heart becomes smaller with an increased stimulation of the accelerator nerve or with an inhibition of the vagus nerve. This can be recognized by an increase in the pulse rate. In his cases, there was an increase of 20 beats per minute, which in itself does not explain the decrease in the size of the heart. Hence, he concludes that thyroid substance acts on the heart muscle itself. He draws an analogy with Kramer's studies of the skeletal muscle in myxedema in which he finds an inhibition. By increasing the tonus there is a decrease in the size of the muscle. In his book, *Die Krankheiten der Endokrinen Drüsen*,²⁰ Zondek attempts to explain the dilatation of the heart and its reduction by two factors: a nervous and a physico-chemical one. He points out that the sympathetic is the tonus nerve to the myocardium and that myxedema is characterized by a high grade of sympathetic irritability, which is seen in the hypotonus of the myocardium which in itself may lead to a chronic dilatation. Thus it leads to serious myocardial injuries, the muscle fibers and connective tissue being filled with an edematous fluid, quite like the mucoid connective tissue of the skin in myxedema. The thyroid stimulates the sympathetic, and the cardiac tissues become markedly dehydrated.

Zandren³⁵ noted the disproportion between the objective cardiac findings and the severe subjective symptoms of insufficiency in certain cases of myxedema heart. In his paper, he discussed Eppinger's theories of edema at length and suggests that the cases described as myodegeneratio-cordis by Eppinger may belong to the same class as the cases of myxedema heart described by Zondek. The results of thyroid therapy accomplished by both authors suggested this to Zandren. He thus explained the mechanism of the thyroid in Eppinger's cases.

SUMMARY

1. Myxedema heart is characterized by an enlargement of all four chambers, slow pulse rate, a normal blood pressure, and electrocardiographic changes.

2. The myxedema heart was first described in 1918 by Zondek, who observed the cardiac hypertrophy and demonstrated the reduction in size after giving thyroid substance.

3. Not all cases reported in the literature are typical examples of myxedema heart. Many authors omit electrocardiographic and x-ray studies. It is possible that certain patients were suffering from coronary artery disease.

4. A case is reported of a fifty-eight-year-old man who presented the typical signs and symptoms of thyroid insufficiency. In this case the teleroentgenogram showed an enlargement of the right and left ventricles. The basal metabolism rate was minus 20 per cent. The electrocardiogram revealed a low voltage in all three leads, a left ventricular preponderance, and a notching and prolongation of the QRS complexes in all leads. After treatment with thyroid substance there was clinical improvement in the myxedema, but no demonstrable changes were observed either in the x-ray outline of the heart or in the electrocardiograms. The second case reported was that of a young man of twenty-six years, whose basal metabolism rate was minus 18 per cent. The electrocardiogram was not characteristic in this case.

5. Many electrocardiographic studies have been made in hypothyroidism. Absent P- and T-waves are well known. Widening and notching of the QRS complexes, and negative T-waves have been described. Low voltages are known to occur. All these characteristics usually disappear after thyroid medication.

6. Theories which explain the above changes are discussed.

7. Methods of safe administration of thyroid gland products and dosage are recorded.

8. There are probably two groups of cases of myxedema heart: those which respond to thyroid therapy, and those in which a long standing myxedema and other factors produce permanent changes in the myocardium which do not permit of a response to thyroid medication.

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Joseph Sailer

IN THE recent untimely death of Joseph Sailer the medical profession has lost a distinguished representative, and preventive medicine, as it relates to heart diseases, one of its staunchest supporters and most active protagonists.

He was among the first to recognize the importance of the public health aspects of heart diseases and early gave his earnest support to the efforts being made to arouse more general interest in measures for the better control of these diseases. It was due chiefly to his enthusiasm and initiative that the Philadelphia Heart Association was organized and became so potent an agent in advancing the welfare of the heart patient. Later he played a leading part also in the organization of the Pennsylvania State Heart Association and was tireless in his efforts to raise throughout that state the standards of the care of cardiac patients. He was one of the founders of the American Heart Association, was an active member of its Executive Committee until a short time before his death and served as its president from 1926 to 1928.

But his interest in the welfare of sufferers from heart disease, intense as it was, represented only a small part of his activities. For many

years as Physician to both the Presbyterian and the Philadelphia General Hospitals and as Professor of Clinical Medicine in the Medical School of the University of Pennsylvania he won fame as a wise clinician and a successful teacher. Upon every phase of his professional work he brought to bear keen insight, sound judgment and a scientifically trained mind.

The world is poorer for the passing of Joseph Sailer and those of us who were privileged to be counted among his many friends have lost a loyal and warm-hearted comrade.

Department of Reviews and Abstracts

Selected Abstracts

Palmer, Robert S., and White, Paul D.: A Note on the Continuous Humming Murmur Heard in the Supra and Infraclavicular Fossae and Over the Manubrium Sterni in Children. *New England J. Med.* 199: 1297, 1928.

Fourteen cases showing the continuous humming murmur best heard on the right side of the neck also heard in the infraclavicular fossae over the manubrium sterni and sometimes on the left side of the neck, have been reviewed in order to call attention to a common physical sign in childhood. In this series of fourteen cases, patent ductus arteriosus was wrongly considered three times and aortic regurgitation was wrongly considered twice.

When the chin is raised and turned to the left this murmur will often be heard in children when otherwise it is not present. It may be heard by this maneuver in some adults.

Although most writers hold that the murmur arises in the veins, this theory is not yet proved. The fact that it is like the murmur of patent ductus arteriosus, the observation that it does, in some cases, persist in spite of occluding the vein, and its great variability in intensity with respiration require further study and observation to determine the exact mechanism of the production. It is possible, as Laennec first thought, that the murmur may arise in the arteries. At any rate the air spaces probably play an important rôle, whatever the vascular origin.

Lawson, George M., and Palmer, Robert S.: Occurrence of Subacute Bacterial Endocarditis in Childhood. *New England J. Med.* 199: 1205, 1928.

At the Massachusetts General Hospital, the earliest age at which subacute bacterial endocarditis has occurred is six years and the next earliest is eight years, a third case is age ten years.

A very unusual case of *Streptococcus viridans* septicemia without evidence of valvular damage in a child twenty-one months is reported. The appearance of the colonies cultured from the blood of this case suggests that it belongs to the subacute bacterial group.

Irvine-Jones, Edith, I. M.: Skin Sensitivity of Rheumatic Subjects to *Streptococcus Filtrates*. *Arch. Int. Med.* 42: 784, 1928.

The author based her study on a varied group of streptococci obtained from the upper respiratory tract of rheumatic subjects and contrasted the findings with the culture group from nonrheumatic individuals. The organisms were classed according to their reactions on blood agar, according to their fermentation tests and according to the skin reactions obtained when filtrates of the strains were injected intradermally into rheumatic and nonrheumatic subjects. Correlation with the Dick test enabled the strains to be differentiated from scarlatinal forms of streptococci.

It was found that the series of organisms from rheumatic sources were very similar culturally and immunologically to those from normal individuals but

that skin sensitivity to filtrates from either series of strains was very much more marked in rheumatic individuals than in the control subjects. This increased sensitivity of rheumatic subjects was more clearly demonstrated with the anhemolytic than with the hemolytic forms.

The skin sensitivity was most marked in subjects during the acute phases of rheumatism, negative reactions frequently occurring as convalescence was established.

The author followed certain rheumatic subjects for a considerable period of time and was able to correlate the changes in the intradermal injection with the clinical condition of the subject and analogy could thus be drawn between her findings and those of Dochez and Stevens on experimental allergy.

The author concludes that rheumatic fever is an allergic manifestation due to streptococci occurring in predisposed individuals. The evidence would point to a heterogeneous group rather than one particular strain of streptococcus as being responsible for rheumatic fever.

Clawson, B. J.: Experimental Rheumatic Arteritis. Arch. Path. 6: 947, 1928.

In this paper a description is given of certain arterial lesions that were produced experimentally. Streptococci had been repeatedly injected into monkeys in an effort to produce glomerulonephritis. Microscopic examination showed in the kidneys of two of the monkeys vascular changes which bore a resemblance to the lesions described by Klotz and Pappenheimer and von Glahn. The two monkeys had received intravenous injections of strains of *Streptococcus viridans*, isolated from the blood of patients having acute rheumatic fever.

In another series of experiments in which many rabbits had had streptococci injected intravenously or subcutaneously in the effort to produce, experimentally, Aschoff bodies and subcutaneous rheumatic nodules, similar examples of arteritis were noted. This arteritis was commonly found in the hearts of rabbits into which streptococci had been injected intravenously, and practically always in the subcutaneous tissues of rabbits into which the streptococci had been injected subcutaneously.

The result of these experiments showed that by injecting streptococci into rabbits and monkeys lesions could commonly be produced in man which morphologically appeared similar to the rheumatic lesions in many. The morphology of the cellular reaction in this experimental arteritis was similar to that found in the Aschoff nodule and in rheumatic inflammation in other parts of the body.

Clawson, B. J.: Experimental Subcutaneous Rheumatic Nodules. Am. J. Path. 6: 565, 1928.

In this paper a microscopic study is made of human subcutaneous rheumatic nodules and of the nodules produced experimentally in the subcutaneous tissue in rabbits by injecting streptococci. The purpose is to compare the structure of a known rheumatic lesion with that of a lesion which has been produced experimentally to arrive at further conclusions concerning the casual relation of streptococci to rheumatic injections.

The nodules were produced in the subcutaneous tissues of rabbits by injecting different strains of streptococci in varying amounts and at different intervals under varying conditions. Five different strains of streptococcus were used. Two of these were isolated from the blood of patients having acute rheumatic fever, one from the blood of a patient with subacute bacterial endocarditis and two from pus from sinuses in cases of sinusitis.

Ten rabbits were injected intracutaneously and subcutaneously in many places with these organisms. Most of the animals had been previously injected

with strains of streptococci intra-arterially through the left ventricle of the heart. Others had been previously injected subcutaneously in one area with a mixture of streptococci and agar.

The reaction found in the injected areas depended on the virulence of the organism, the number of organisms injected and the time of the removal of the nodules after injection. The most virulent organisms tended to produce local abscesses, also if the area of injection was excised shortly after injection, there were signs of abscess formation. If organisms of low virulence were used or if small numbers of organisms were injected if the area of injection was excised at a later day there were found typical proliferative nodules at the site of injection. The reaction observed in these nodules was similar to those found in the nodules removed from human beings. Since these experimental nodules occur obviously as a result of injected streptococci, the probable conclusion is suggested that acute rheumatic fever and the type of inflammation associated with it are of streptococcal origin.

Kreidler, William A.: Biologic and Serologic Studies of Streptococcus Cardioarthritidis. J. Infect. Dis. 43: 415, 1928.

The author has studied the biologic and serologic reactions of a series of 107 strains of this organism. Three strains were obtained from blood cultures, three from cultures of feces and the remainder from throat cultures. All the strains fermented glucose, sucrose, inulin, salicin and raffinose; none, mannitol. Twenty-one strains failed to ferment lactose and failed to produce acid in milk. None liquefied gelatin or produced indol.

Antigens prepared from each of the strains were agglutinated by a monovalent antiserum of *S. cardioarthritidis* in dilutions high enough to indicate that these strains fall into a definite serologic group and that group agglutinins play but a small part in the results obtained.

When the foregoing facts are considered, there seems to be reason for the belief that these microorganisms biologically and serologically form a compact species of streptococci. The failure of some of the strains to ferment lactose and the difference in the agglutinability of the strains, suggest that there may exist, within the species, immunologic subtypes of this organism.

Belk, William P., and Fendrick, Edward: The Lesions in Animals Inoculated with Streptococcus Cardio-Arthritidis. Arch. Path. 6: 812, 1928.

This report is based on the anatomic lesions found in seven rabbits and two horses inoculated with repeated injections of *Streptococcus cardioarthritidis*. The animals were given viable twenty-four hour cultures suspended in saline solution. The lesions in the various tissues of the body resembling those seen in human rheumatic fever were identified. These lesions are described in the paper.

Epicarditis appeared in two of the rabbits; nonpurulent focal myocarditis in six; mural endocarditis in three; valvular endocarditis in four; arteritis in four; myositis in three; bursitis in one; glomerular nephritis in one; infarcts of spleen carditis, focal myocarditis, valvular endocarditis, arteritis and pneumonitis ap and kidney in one; aortitis in three and pneumonitis in all the animals. Peri appeared in both horses.

Poynton, F. John: Rheumatic Heart Disease in Childhood. Lancet 215: 537, Sept. 15, 1928.

In these 3 lectures, the author discusses three particular phases of rheumatic heart disease. First, the correlation of the pathological lesions of the heart

with other rheumatic lesions; second, the clinical manifestations of rheumatic heart disease and third the treatment of heart disease.

The author describes the two chief ways in which acute rheumatism damages the heart and other organs, assuming that a streptococcus is the cause of the disease. In the first process there are the local interstitial lesions which tend to necrosis and fibrosis and in the second process there are the toxic effects of the circulating poisons. The interstitial lesions produce results which clinical observation at the bedside corroborates: valvular lesions, pericarditis and local myocardial disturbances. The toxic effects of the infection though well recognized at the bedside in the profound anemia and damage to the cardiac muscle and nervous system cannot yet be correlated with any definite poison. The author regards chorea as a rheumatic meningo encephalitis.

Campbell, John S.: Stereoscopic Radiography of the Coronary Circulation. *Lancet* 215: 168, July 28, 1928.

The author has devised a method of infusing human hearts for roentgen examination. A preparation of barium, Rontyum has been found to give the best results. This is injected in the vessels of the heart under a pressure of 280 mm. of mercury maintained for three minutes. After injection of both coronary arteries, the chambers of the heart are packed with wool and the organ immersed in 10 per cent formalin for twenty-four hours to secure fixation in the best position for stereoscopic examination. This simple method has been found to give satisfactory pictures.

The skiagrams demonstrate very clearly the finer ramifications of the cardiac vessels while small variations in their caliber can be readily noted. The variations in the distribution of the coronary vessels noted by Gross have been confirmed but their occurrence has been greater in this present series of 92 hearts examined by the author.

The alteration in the relative vascularity of the ventricles which comes with advancing age and results in a decrease of the blood supply to the right heart through an increase in the development of anastomotic branches was also verified. Diminishing vascularity of the heart similar to that found in senility was found to accompany coronary disease.

Removal of portions of the ventricular walls made possible a stereoscopic study of the septal circulation and the supply of the neuromuscular tissues.

Whitten, Merritt B.: A Review of the Technical Methods of Demonstrating the Circulation of the Heart. A Modification of the Celluloid and Corrosion Technic. *Arch. Int. Med.* 42: 846, 1928.

The author reviews the literature pertaining to methods used in preparing casts of the interior of the heart and of the coronary circulation. He also described his own technic for preparing specimens using celluloid as the material for forming a cast and the corrosion method for destroying the heart tissues. The left coronary arteries in the completed specimen are injected in red, the right coronary arteries in blue and the coronary veins in white. The wall of the heart is destroyed and the background of the vessels is the white cast of the chambers of the heart, depicting the internal surface of the heart area.

Dumas, A.: Hypertensive Form of Mitral Endocarditis. *Presse Med.* Sept. 15, 1928.

The author finds that 9 out of 40 cases of chronic endocarditis which were confirmed by autopsy were accompanied by hypertension. He notes that this association is commonest in subjects over 50. The mode of death in 6 of the 9

instances was by progressive weakness and failing tension. In 3 of the cases death occurred more rapidly with edema of the lungs and in these cases the heart showed very little hypertrophy.

The clinical findings in such cases are of pure hypertension. No nephritis was present and a few old infarcts were found which could be due to the heart condition. Little cardiac hypertrophy was present and albuminuria was only an occasional finding.

The etiology of the mitral disease is doubtful. In two cases, acute articular, rheumatism had been noted in the history and in one instance syphilitic aortitis was present.

The physical signs of the heart are generally those of mitral insufficiency although autopsy shows definite stenosis.

In some cases a presystolic murmur was heard and was confused with the gallop rhythm of a hypertensive heart. In some subjects the lesion is silent.

The author considers the mitral endocarditis as responsible for the development of the mitral stenosis.

Herrick, James B.: Treatment of Heart Disease. J. A. M. A. 91: 1761, 1928.

In this address the author apologizes for a very simple, old-fashioned paper. He points out that plain truths have to be repeated or presented in forms to fit the passing moment. He discusses the all-important subject of rest and digitalis, pointing out the difficulties in determining in each individual patient just what degree and kind of rest and medication are necessary. He believes there is no hard and fast rule for these two phases of treatment.

Hepburn, J., and Graham, Duncan: An Electrocardiographic Study on 123 Cases of Diabetes Mellitus: Am. J. M. Sc. 176: 782, 1928.

It has been shown that in a series of 123 diabetic patients 56 showed serious electrocardiographic abnormalities at the beginning of the diabetic treatment and that in a very fair percentage the electrocardiogram returned to normal after the diabetic condition was controlled by treatment. It would appear from these observations that in the cases in which the electrocardiograms returned to normal after the diabetic condition had been controlled, that the abnormal electrocardiograms had resulted from the effect on the myocardium of the perverted metabolism present in diabetes mellitus. The authors discuss whether these changes might be due to a direct action on the myocardium or an indirect one resulting from disease of the coronary artery. None of the few cases of severe acidosis studied showed an abnormal electrocardiogram and many severe diabetics with hyperglycemia had normal electrocardiograms. They conclude that diabetes mellitus apparently does not produce any direct effect on the myocardium resulting in an abnormal electrocardiogram. The changes in the electrocardiogram found in this study resembled those seen in nondiabetic patients suffering from cardiovascular disease and are supposed to result from the indirect effect produced on the myocardium from disease of the coronary artery.

Lemann, I. I.: Coronary Occlusion in Buerger's Disease (Thromboangiitis Obliterans): Am. J. M. Sc. 176: 807, 1928.

In thromboangiitis obliterans the pathological process is probably not limited to the vessels of the extremities. Affection of the coronary vessels has been reported in three cases collected in the literature. The author reports another case showing extreme arteriosclerosis. The lesion of thromboangiitis obliterans was not present in the coronary artery.

Bromfin, I. D., and Simon, Saling: Observations on Some Cardiac Lesions Coincident With Pulmonary Tuberculosis. *Am. Rev. Tuberc.* 18: 727, 1928.

The incidence of valvular heart disease in pulmonary tuberculosis recognizable clinically is only about 6 per cent in the experience of the authors. Dyspnea out of proportion to the pulmonary involvement especially when there are no constitutional symptoms should arouse the suspicion of an existing cardiac affection.

Artificial pneumothorax when indicated in such cases should be administered with great caution. The earliest manifestation of cardiac disturbance is an indication for discontinuing the pulmonary compression.

Complete bed rest must be rigidly enforced for a longer period of time than in cases not complicated by heart disease.

The electrocardiogram is often of value in determining the cause of obscure cardiac symptoms. The authors found an instance of bundle-branch block in a patient without symptoms indicating such a disturbance of mechanism.

Starr, Isaac Jr., and Gamble, C. J.: An Improved Method of Determination of Cardiac Output in Man by Means of Ethyl Iodide. *Am. J. Physiol.* 87: No. 2 450, 1928.

The authors have modified the ethyl iodide method of Henderson and Haggard for the estimation of the cardiac output. In their earlier papers they showed that the coefficient of distribution of ethyl iodide between air and blood as originally expressed by Henderson and Haggard could not be confirmed, neither was the ethyl iodide destroyed in one round of the circulation as was originally thought.

By continuing a modification of this method with the Fick principle, the authors have obtained reliable results in men.

Several technical points stressed by the authors include:

1. The estimation of the ethyl iodide in air by the precipitation of silver iodide in the distillation of the blood previous to determination. The authors use capryl alcohol to reduce the pressure.

2. The amount of blood used has been raised to 60 c.c., thus permitting a reduction in the concentration of the ethyl iodide used.

3. The distribution coefficients of the blood previously reported by the authors to average 7.6 is, they believe, lower as with bigger samples it averaged 6.1. The presence of fever or of anemia lowered this figure and the figure for dogs is higher, averaging 10.7.

The authors demonstrated in dogs and in human subjects that the arterial ethyl iodide may be estimated from that of the alveolar air and that the ethyl iodide content of mixed venous blood may be estimated from the re-breathed air.

They were able to get consistent figures for blood flow in dogs by the ethyl iodide method when the lungs were perfused at a known rate.

A description of various manifestations of the apparatus is given with consecutive determination on two subjects. The method requiring no active co-operation from the subjects employed.

Eppinger, H., Lazlo, D., and Schürmeiyer, A.: On the Probable Causes of Waste of Energy in the Organism with Heart Failure. *Klin. Wchnschr. Jahrg.* 7, No. 48, 2231, 1928.

The authors have correlated the problems of hemodynamics with those of metabolism. Patients with cardiac decompensation use more oxygen in relation to their size than normal individuals and it is found that after exercise an

excess of lactic acid is present in their blood. This latter points to faulty muscle metabolism during work with failure of reconversion into glycogen. That this is evidently not entirely due to a deficient supply of oxygen is shown by the fact that the arterial oxygen is generally normal in amount. It is suggested that the explanation may be similar to that described by Eppinger as occurring in shock and collapse; namely, an altered distribution of the blood between circulation and tissues, so that the arterial blood volume and hence the supply to the muscles is decreased.

In histamines and more especially in peptone shock and failure in blood pressure occurred and the fatigue curve of the muscle corresponded to this; as the blood pressure rose the fatigue picture disappeared.

To show the effect of local change in circulation the authors ligated the femoral artery and vein ten minutes. Only minor changes in the muscle were observed while ligation of the aorta lead to changes resembling those of shock. Ligation of the vena cordis did not produce any marked change. Bleeding and suffocation produced rapid fatigue, but no change unless the oxygen was reduced below 8 to 10 per cent.

The lactic acid of muscle and blood under the above-mentioned conditions was studied and it was found that during histamine and peptone shock and after ligation of the aorta that the lactic acid was increased in the muscle. Decrease of oxygen in the air did not alter the lactic acid as long as the minute volume could be increased.

The authors consider that the failure of resynthesis of lactic acid into glycogen as due to circulatory insufficiency with oxygen lack in the muscle.

In narcotized dogs studied from the standpoint of shock and ligation of the aorta with and without work there was an increase in the oxygen consumption due to a diminished resynthesis of lactic acid into glycogen.

To demonstrate the effect of shock on the blood supply to the muscles, the author observed a cat muscle under the microscope using Krogh's technic. Histamine produced a stasis in arteries and veins and agglutination of the red blood corpuscles. The effect of muscular work on the minute volume in histamine shock was also observed and a decrease noted. The volume of circulating blood was not estimated but it is assumed from previous observations that in decompensation considerable amounts of blood are deposited.

Thus the authors believe that capillary damage leading to deposition of blood and diminution of the blood volume occurs in decomposition. This results in a diminished oxygen supply in the tissues, so that lactic acid is not reconverted into glycogen.

Eppinger, H., and Hinsberg, K.: On the Possibility of a Peripheral Treatment of Patients with Heart Disease. Klin. Wehnschr. Jahrg. 7, No. 48, 2284, 1928.

From the foregoing paper, the authors draw certain conclusions for the treatment of heart disease and suggest massage as well as the usual drug therapy. They show in three patients with chronic heart disease that a general massage of the muscles leads to a reduction of the oxygen debt and the oxygen consumption during work. Besides the objective findings, the subjective conditions of the patients were improved. They recommend the use of massage in patients with chronic heart failure after the stage of decompensation.

Eyster, J. A. E.: Experimental and Clinical Studies in Cardiac Hypertrophy. J. A. M. A. 91: 1881, 1928.

The increase in the muscle mass of the diseased heart occurs mainly in that part of the heart placed under a mechanical handicap as a result of a

lesion. The theory of work hypertrophy has been almost universally accepted in explaining this increase in size as a result of the increased activity and work necessary to overload the mechanical defect.

It has been pointed out by Albrecht that in human hearts the seat of myocardial disease, the stimulus to hypertrophy may be impaired nutrition resulting in work hypertrophy. The author has pointed out previously that dilatation of the heart muscle precedes hypertrophy in cardiac lesions and in several types of experimental lesions produced in dogs he has shown by roentgenologic methods that a period of increase in heart volume occurred immediately after the lesion was produced associated with macroscopic and microscopic evidence of stretching and injury to the muscle. This initial increase in volume disappeared after a few days and the heart returned to its normal volume or even below. Subsequent to this transitory period of stretching and dilatation, a second and more gradually developing increase in heart volume occurs which is due to increase in muscle mass or hypertrophy. This is completed as the result of a single lesion in approximately one hundred days. A second lesion, superimposed on the first causes a second period of transitory dilatation and the subsequent period of additional hypertrophy.

As a result of these studies, the author proposes a new theory as to the cause of heart muscle hypertrophy. This theory in contrast with the theory of work hypertrophy may be designated as the theory of injury hypertrophy and he ascribes increase in muscle mass not to a physiological response to increased work but to a tissue response of some nature to actual injury. During the period of stretching the muscle fibers are the seat of a process of hydropic degeneration, a typical injury and reaction to injury.

In order to test this matter further temporary cardiac overload was produced in a series of dogs by massive transfusions. The estimated blood volume was increased from 75 to 100 per cent in a series of 11 animals. These have shown an initial dilatation comparable with in every way to that produced by aortic lesions, except that it lasts for a somewhat shorter period. During this period the ventricular muscle shows similar macroscopic evidence of stretching and injury. The heart volume then returns to near its normal or even below as also occurs after aortic lesions, and finally shows a second more gradually developing increase in volume due to hypertrophy of the muscle. A second massive transfusion leads to a repetition of the whole process, a second period of dilatation being succeeded by a second period of additional hypertrophy, comparable again to the production of a second experimental lesion superimposed on the first.

Estimating the heart size by the use of x-rays and certain other methods of physical examination, the author has studied 70 men and 40 women forming a group of university athletes. The author believes that in absence of cardiovascular disease that cardiac hypertrophy in man does not exist.

He concludes from these experimental and clinical observations, that the most important factor leading to the cardiac hypertrophy that develops in organic cardiac or vascular disease is not increased work of the muscle per se but the muscle injury and the reaction to injury that results from abnormal stretching of the muscle in the initial period of overload as the lesion develops.

